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The Arthur E. Mills Memorial Oration.[1]

THE SECRET OF LIVING.

By S. W. PENNYCUICK, D.Sc.,
Reader in Chemistry, University of Adelaide.

IN the year 1500 there lived in Europe a strange personality. His name was Phillipus Aureolus Theophrastus Bombastes von Hohenheim. He was a physician: a doctor of medicine. He had been an army surgeon in the Danish wars. When appointed city physician in Basel he signalized his appointment by publicly burning all the medical books in the library to show his contempt for his contemporaries.

He was also a chemist, an accomplished experimental chemist. In fact, it was he who gave us the word alcohol: he changed spirits of wine to alcohol of wine, and it has been alcohol ever since.

He left many books on medicine and chemistry. Those who read his books declared, not without reason, that Aureolus Phillipus Theophrastus Bombastes von Hohenheim wrote only when in a state of intoxication.

He was indeed a curious personality—in part, an outrageous mountebank, a drunkard, noisy, boastful and quarrelsome, given to the most violent and abusive invective, the like of which is not heard amongst scientists today—unfortunately. And yet, he was an outstanding physician and chemist. The chemists, by the way, have handed him over to the physicians, and the physicians have handed him back again. So that today he goes down in history as a chemist, known by the less exalted name of Paracelsus.

The major claim to fame of this strange man was, first, that he succeeded in rousing the physicians from their state of self-satisfied somnolence, and second, he succeeded in raising the chemists from their state of alchemistic degeneracy.

"Leave the transmutation of the elements alone", he said to the chemists. "That alchemistic rubbish gets you nowhere." And to the physician he said: "Why don't you try and find out something about these crude mixtures of herbs and barks and roots and leaves and flowers that you handle, and about which you know nothing?" And to both he said: "Work together! Experiment! Medical preparations must be handled by artists. Let the chemist be the artistic handmaid of medicine."

And since that day, right through the centuries, although chemistry has become a mighty science touching all things upon this earth, it yet remains in some degree the handmaid of medicine, preparing, purifying and synthesizing new drugs.

Maybe, it was with this handmaid idea in mind that tonight I, a chemist, have been called forth by my masters to deliver this address—this Mills Memorial Oration, founded in honour of the late Professor Arthur E. Mills of Sydney, ex-Dean of the Faculty of Medicine, ex-Deputy Chancellor, a Foundation Fellow of this College, who was a great physician, a great teacher and a great administrator. Tonight we pay him homage.

In keeping with his greatness, I have chosen a great subject, or at all events a great title: "The Secret of Living." Maybe it will be a greater secret than ever by the time we have finished

[1] Delivered at the annual meeting of The Royal Australasian College of Physicians at Adelaide on May 29, 1952.

with it tonight. It is an impossible subject, you will say; a hopeless task; only a brave man would attempt it. I agree. Only a brave man would stand here and give an oration.

But first, let us follow this master and handmaid for a moment. The physician said to the chemist: "For centuries I have been using opium as an opiate—a sleep producer. But opium as it comes to me is a crude product, varying from sample to sample. Cannot you purify it further? Or better still, could you isolate the all-important chemical that it contains?"

Now this was a difficult problem, for opium is a complex mixture, containing a dozen different alkaloids. But the chemist set to work and in the end he produced pure morphine, which exists in opium to the extent of roughly 10%. One man alone, a chemist named Serturner, spent fourteen years of his life upon this problem, and he was instrumental in handing it to the physicians, 100% pure. What a blessing pure morphine has proved in the physician's hands! Serturner, by the way, died a painful and agonizing death, for he was one whose stomach rejected morphine. This, of course, was before the days of the hypodermic syringe.

The physician said to the chemist: "I wish you could improve the local anæsthetic, cocaine. I know you say it is quite pure, but unfortunately it has some unpleasant after-effects and it is somewhat toxic. Can you do anything about it?"

So the chemist set to work. He took the cocaine molecule apart, then rebuilt it on a different plan, introducing dozens of variations, getting better and better products, more effective anæsthetics and yet less toxic, like "Holocaine" and "Stovaine", until at last he got "Novocain", sold as procaine or procaine hydrochloride today, which was exactly what was wanted.

Or again, the physician said to the chemist: "For a thousand years bacteria have been the bugbear of my existence. Every year they bring misery and death to millions of human beings. Cannot you find something that will help to kill them in the human body?"

Now here indeed was the most baffling problem of the ages, the most baffling chemotherapeutic problem. The chemist knew scores of things that would kill bacteria; but what would kill the bug would kill the human—for men and microbes are strangely alike. Then after he had almost given up hope, only sixteen years ago—only sixteen years ago, mind you—came the thing that chemists had been looking for for centuries: sulphanilamide. It was not a natural product; the chemist built it for himself in his laboratory. Having got the clue, he played with this molecule as few molecules have been played with, before or since. He took it to pieces and rebuilt 5000 different variations—5000 sulphonamides—all built to careful specifications. From these, with the help of the physician, he chose the best, and thus we have "M. & B. 693", sulphapyridine, sulphamerazine, sulphadiazine and all the famous "sulpha" drugs. Their importance in medicine today can scarcely be overestimated.

All these things, and a thousand more, the chemist produces with artistic skill and great patience. For Paracelsus was right: the chemist is an artist. To paint the picture of a molecule of penicillin cost perhaps £1,000,000, and it took three years of teamwork before the artists were satisfied they had got it right.

And what do chemists get out of all this protracted labour? Make no mistake, they get great satisfaction—the satisfaction of a job well done. Maybe, they get a little honour and glory, *plus*, of course, a precarious livelihood. They also get the knowledge that they are helping physicians in their great task of keeping people healthy and keeping them alive. For perhaps the greatest desire of the human being is first to keep physically well (which is understandable), and second to keep alive (which is even more important).

Now, why should a man die when his cells can live forever? The cells are immortal, it is the organization that fails. Speaking very generally, men and women would live forever if they could avoid three things: accidents, bacteria and chronic diseases. We could call it the A B C of life. A—accidents, B—bacteria, and C—chronic degenerative diseases. Avoid these and never die. It is as simple as that.

Then why don't we do it? It is worth a moment's attention. With accidents we include all forms of violent death; although in these times of merciless war all violent deaths are not accidents. There is the story of the young man from the country who went to a city physician for a life insurance examination. "Have you had any accidents?" the doctor asked as he wrote down his details. "No! No accidents", said the young fellow. But presently, when the young man took off his shirt, there was a huge scar across his chest. "What's that?" said the doctor. "Oh", said the young man somewhat proudly, "a bull did that." The doctor was annoyed. "You told me you had never had an accident." "It wasn't an accident", said the young fellow, "the bull did that on purpose."

Quite seriously, accidents and violent death are a major problem in the world today. In England, for every death on the road there is an accidental death in the home. In America, accidents are rated as the chief cause of death among children between the age of one and fourteen years. In Australia, about 300,000 factory accidents are reported each year. On top of this, all over the world in the last forty years, millions upon millions have been killed in war by cruel design; and as far as one can judge worse might follow. Our boasted progress seems to have got out of hand. Perhaps this business of keeping alive is not so simple after all.

But when we pass to B—bacteria—the story is entirely different. Throughout the centuries, up to about 100 years ago, the microbes were the masters. Against them doctors fought a losing battle. Only the fighting power of man's own leucocytes saved the human race from extinction.

Then, about 100 years ago, came Pasteur, then Lister, Koch, the germ theory, disinfectants and so forth. Then, only sixteen years ago, came the first of the modern chemotherapeutics, the sulphonamides (as I have mentioned), quickly followed by penicillin and the other antibiotics, streptomycin, aureomycin, and the like. The result is that today the physician is fighting the microbe on better than even terms; in many ways he has the upper hand. The bugs of course are fighting back, but it is a sort of rearguard action.

This has been a great triumph for medicine: millions of people are now kept alive; the average span of life has gone up with a jump; and, incidentally, fresh economic and social problems are gradually leading to a tremendous crisis. But that is another story.

Having, by the grace of God, successfully avoided accidents and violent death, having with the help of the physician fought off the insidious little microbes, man, in this A B C of life, has then to face the third and final hurdle—the final hurdle, for this is the one that will ultimately bring him down—the chronic diseases. The chronic degenerative diseases. They are not caused by microbes, nor are they infectious. Usually, but not always, they bide their time and take their toll with the advancing years. Diseases of the heart, rheumatism, arthritis, cancer, nephritis, arteriosclerosis, mental disorders, diabetes and other glandular disturbances more or less obscure.

Too often they are the signal that the body is wearing out—the extraordinary complex mechanism is beginning to crack. Doctors of course can hold them in check, or ward them off with great success. You know the old saying: "If you wish to live to a ripe old age, get a chronic disease when you are young—and look after it."

But in the end, no matter what precautions we take, one of these troubles will cause the body to falter, then stop.

Medical science has accepted the challenge. More and more the hormones are coming into the attack—thyroxin, insulin, and now cortisone and ACTH; others are sure to follow. When the hormone mysteries are sorted out, when the secrets of the enzymes, the vitamins, the anti-enzymes, the anti-hormones and the anti-vitamins—when these secrets are laid bare, when the protein problem is solved and the chemical handmaids have provided a thousand new drugs, then undoubtedly life will be prolonged still further.

How far? A hundred years? Some say 150 will be the normal span of life. I doubt it. Not unless some of the internal organs are completely redesigned. It may well happen that some electronic genius, for instance, might one day design a special biochemical-electronic organ, say a heart or a liver—a substitute, an external one—one that people can push round with them on a barrow.

But who would want to live then? Imagine what things would be like with three-quarters of the people living on the old-age pension (pushing their barrows round) and the other quarter living on child endowment. What a time the economists would have!

However, there is one thing that is certain, and that is the desire to live dies hard. And from what has been said so far

tonight one might draw the conclusion that the "Secret of Living"—the subject of this address—is to be found in the A B C of life; avoid accidents, bacteria and chronic diseases, avoid them as long as you can. But that is not the secret of living at all.

I must confess that I have somewhat wilfully brought you along this wrong path in order to stress how easy it is for human beings to concentrate upon the material side of life. We do it all day long. One of the great faults of this modern scientific age is its over-emphasis on material things and material values. Only those things are real which can be measured or demonstrated by experiment; all the rest is hazy. That, unfortunately, is what the world seems to be gleaning from the teachings of modern science.

Lord Kelvin says somewhere: "When you can measure a thing and express it in numbers you know something about it, but when you cannot measure it or so express it your knowledge is of a meagre and unsatisfactory kind."

This may be strictly true in the realm of science, but it is not true in this business of living. The important thing in life is not the exact number of years we pass on earth, which can be measured to a fifth decimal place, maybe to a fraction of a second (I hope the obstetricians agree)—the important thing is how we live those years. There are all sorts of things in our daily existence which cannot be measured or expressed in figures or cured by drugs or by the surgeon's knife, and yet which have a profound influence on our well-being and our happiness.

Avoiding accidents, bacteria and so forth—that is not living, that is merely keeping alive. Living, getting the most out of life, and what is equally important, putting the most back into it, is a different matter altogether.

Bernard Shaw, in a characteristic caustic epigram, says that many people should have on their tombstones the epitaph: "He died at thirty, he was buried at sixty." Or to put it in another epigrammatic form: he lived only half his life.

The point is only too clear.

Well, then, wherein lies the secret of living? Wherein lies the elusive secret of getting the most out of life and putting back as much as we get?

It is strange how people seldom ask themselves the basic question: "What do I really want from life?" "Why do I strive and struggle and worry day by day?" "What am I after?"

The modern young person might say, rather crudely but succinctly, that there are two things in life: getting a living and having a good time.

At first blush that sounds rather awful; but except for its crudity it is not very far from the truth. If we put it less crudely—and even more succinctly—and say, the two important things in life are work and happiness, then, allowing for the extraordinary economy in words, that is not a bad summary of life.

In effect it says there are two sides to life, the material side represented by work—we all have to work, it is our daily preoccupation—and the non-material side represented by peace of mind, contentment, happiness, call it what you will. The two of course are complementary; but for the moment let us keep them apart. Let us assume that there is a material side of life and a non-material side.

On the material side there are of course other things besides work. There is good health, for instance. Everyone wants good health, although some academic people argue that poor health has its advantages, it sharpens the mind; they quote exceptions from history to prove it. But for my part, like most people, I prefer to be dull and healthy.

Then of course everyone wants to keep alive, or nearly everyone; it is the first law of Nature. However, we have already overstressed these things; we shall now leave them to the doctors, where they belong.

Outstanding amongst the other material desires are the need for security, freedom from want and to a lesser extent freedom from fear. Man needs these things for himself, his wife and his children. To get these things he goes out to work; for work means money, and money buys these things.

It is a platitude of course that money does not buy everything. But let us not pretend to undervalue it; by our social-economic system money does buy security, food, clothing; without it we would starve. But we must be very careful not to overvalue money; for this is perhaps the commonest individual error in the world today. Money simply does not buy the best things in life.

It is not for money that we really strive; it is for security, freedom from want, and so forth. In fact, insecurity, downright fear and want are the great driving forces that get things done; they are the driving forces in the struggle for existence of all creatures on this earth, big and small. Without this driving force all creatures, including man, seem to be at a loss, and life becomes empty.

The animals in the zoo have 100% security, complete freedom from fear and want, but they seem to pass from restlessness to discontent and then to utter boredom.

And if some unfortunate man were rounded up by the animals and put in one of their zoos (for educational purposes; labelled Genus *Homo*) on public holidays the other animals would gather round, some would say: "What is the matter with him? He has security. He is completely free from fear and want. He has everything: free medical attention. In fact, he has the 100% welfare State. Why does he look so beastly bored and discontented?"

Others would say: "Why don't they let the poor thing out?"

But the wise ones would say: "It is not security, food, freedom from fear and want that that creature really desires; it is the striving for these things that brings him pleasure and makes him happy and contented. The funny thing is he does not know it. That is why we have him in the zoo. Read the label on his cage—'Genus *Homo*: Species *Sapiens*': 'The creature with superintelligence, but the only creature in existence that does not know what it really wants'."

That brings me to the first of the secrets of living.

Security, freedom from fear and want and the money that buys these things, substantial as they seem, they are but the shadows, for once we grasp them they seem to disappear. The substance is the happiness that comes with striving. If work and happiness are the two big things in life, then finding happiness in one's work is surely the first and foremost of the secrets of living.

That holds for everyone, men and women alike, in all degrees and stations. For everyone must work—work or perish. And although we claim to protect our womenfolk, many women nowadays seem to work harder than the men.

But no matter whether man or woman, no matter the degree or station, there is joy in work that can be replaced by nothing else.

Those people who find it easy and natural to become absorbed in their work—physicians, for instance, and millions of others—they have a tremendous advantage in life. Many women, too! I heard a woman say one day: "What time have I to look for happiness? I have four children and no help." What need had she to look for happiness with four young lives to mould? Strange how people think they have to chase happiness in the highways and byways when all that is needed is to recognize it wherever it lies, so that one can pick it up. Time will show that the happiest years of one's life were the years when one was most busy—too busy to notice it. That is why we put work and happiness together.

Those men and women, high and low, who look upon their daily work as a sort of necessary evil, something that has to be done, or something that you get out of if you can—they are terribly handicapped for they are throwing away the very first secret of living.

And those people who deliberately discourage others from taking a joy in their work—well, they are just wicked. For wherever one finds a tendency to discount work as a major contribution to a happy life, wherever people are encouraged to be slipshod, to get as much as they can and give as little in return, where pride and satisfaction in work are discounted and money is set up as the real objective in life—there you will always find dissatisfaction, discontent, unhappiness. Psychologically it is wrong; for one's happiest hours are to be found in one's daily toil and in a job well done.

So we shall mix work and happiness together as the first of the secrets of living.

And what are the other secrets? Are there any more? Plenty.

Life is a complex mixture; rather a glorious mixture. Let us proceed with the analysis.

If living means getting the most out of life and giving the most to it, then giving and getting, like work and happiness, must be complementary. Just as a nation cannot get more out of its national life than it puts in—exports must balance imports, standard of living (what we get out) must be balanced by production (what we put in)—so the individual must keep a balance between what he gets out of life and what he puts in.

But the troubles that Prime Ministers experience in striking an import-export balance are nothing to the troubles we individuals have in striking our give and get balance; for the scales are weighed against us all the time. Right through life we are taught to concentrate on getting, on acquiring things. A man's success in life is measured by what he gets. The most overworked verb in the English language is the verb "to get".

We get attention in the cradle, we get presents, later we get an education, we get a job, we get a living, we get married, we get a house, we get a motor car—and in the end, we get a funeral. And what do we give in return? Precious little, when it is all balanced up. And yet, there is more real joy and happiness in giving than in getting.

To take a homely example: How often per year does a man give his wife an unexpected present? Suppose the spirit moved him, and suppose one day a man did bring home an unexpected present—mind you, something his wife had been asking for for six years, but still unexpected—what would her reaction be? First surprise and incredulity, then joy, and then, I am afraid—a slowly dawning suspicion.

That is what it has come to. Giving freely, as a habit, without thought of return, recompense or reward, just for the joy of giving, is a rare and precious thing. Mind you, there is plenty to give: service, love, loyalty, devotion, *plus* something unexpected now and then, big or small. The really happy people are those who have the gift of giving.

It is such a precious gift that we shall place it right up on the list of the secrets of living.

Now it is by no means surprising to find in an analysis of life that the non-material things play a major part in bringing contentment, peace of mind, satisfaction, or, for want of a better comprehensive word, happiness.

There is, for instance, the spirit of tolerance that is so lacking in the world today: tolerance, understanding, forbearance, they all go together; and with them goes that magic domestic word "consideration".

It is intolerance and lack of understanding that so easily lead to wars big and small. It is intolerance and lack of consideration that so easily lead to domestic wars big and small. Look how easy it is to walk into a quarrel when both sides are intolerant. And yet, there is an inner satisfaction to be found in treating our fellow beings with forbearance and understanding, meeting them more than half way.

To take a homely example: If a woman had a difference of opinion with her husband, what should she do? Let her (after cooling down) approach not half-way but two-thirds of the way towards him and hold out her hand. For it is a strange mathematical anomaly that in any disputation two sides will never meet if they advance half-way towards one another; to meet they must advance at least two-thirds of the way towards one another. If he does not move, let her then approach three-quarters of the way; if he still does not move, let her approach seven-eighths of the way towards him and hold out her hand. If he still does not move, well, if he is that sort, then the rules no longer hold and at least she will be in a beautiful position to strike him if she so desires.

Which means, of course, that we are all human, and our ideals are always a little beyond our reach.

Nevertheless, in this somewhat discontented world we must put tolerance and forbearance, understanding, consideration on our list of the secrets of living.

Life is indeed a complex misture. Like a good Paracelsian handmaid I have sought to analyse it tonight, to distil some of its secrets and to present them to my masters.

There is one more that I would put before you. It is big. It is non-material. It can be summed up in the little word—humility. Those who understand so much realize how little they really know. There are so many things the human mind simply does not understand, things that seem beyond the reach of reason, that it is befitting that we should be humble.

And with humility goes faith. Without faith people, strange to say, have nowhere to put their feet.

No matter how some may argue, there has been no single life ever lived on this earth that has so influenced the lives of man as that of the founder of Christianity. The message that it brought to men and women has not altered in two thousand years; and that message is the Christ-like way of life—faith, humility, mercy, justice, understanding, love, forbearance.

Scientists and the great experimenters of the world can find out what they will about hormones and antibiotics, genes and enzymes, about the secret of the stars, about the expanding universe and the galaxies that slip forever across the horizon; they can develop new ideas about continuous creation, how the earth was formed and how life began—but human beings will still feel that somehow something is lacking. The plain man and woman will still long for a world of Christ-like ideals as they have for twenty centuries past.

Science cannot fill this gap. Education cannot bridge it. Orthodox medicine stands helpless before it. To plagiarize: What know they of medicine who only medicine know? Man is not just a machine. He is an active being with power to direct his strivings towards an ideal goal.

And in our search for the secret of living tonight we have been led to the ideals for which man strives. We are not looking for the perfect man or woman. We are not interested in perfection. We are looking for those things that make life worth while. And we have found them in four simple rules. There is nothing negative about them; no don'ts and forbiddings. Be humble and full of faith, be tolerant and understanding, cultivate the precious gift of giving, find enjoyment in your daily work. It is all positive and deceptively simple.

But in practice, how difficult it is. Or put it the other way round, look how easy it is to break some or all of the rules—to be intolerant and overbearing, to be without faith of any sort, to be continually on the make, grabbing and greedy, or, to be downright lazy. We break the rules all day long. Somehow human beings seem to be made that way; it seems as though we were not made to be happy. And yet the rules are there all the time. Mind you they are not new; they are as old as the hills; but they will be new until eternity. For each man and woman must learn anew that happiness comes with striving.

To travel hopefully is better than to arrive. As a boy I thought that saying the most foolish on earth; for what is the use of travelling anywhere if you don't want to get there? Far better to stay at home. True enough, the saying is quite inappropriate when applied to a transport system. "To travel hopefully is better than to arrive" would be sadly out of place on the walls of our railway carriages, for instance. It would give rise to very nasty comments.

But life is different. In life the destination is not written on the ticket. Our journey through life has little meaning unless we travel hopefully towards the ideals which we know full well we will never reach.

One becomes disturbed at the directionless travelling in life today. Maybe science has done its job too well and concentrated thoughts too much upon material things and upon material comforts. For many years I have taught thousands of students the value of exact measurement, of careful experiment, of adding to the store of facts—in short the value of material things. And I have watched with some dismay the ascendency of the material mind. So sometimes directly, but always indirectly, I have sought to show that life is not the summation of material things; there are values that cannot be measured or analysed or examined with a microscope.

And tonight, in maybe this sort of swan-song, I bring this message to you. Have faith in your ideals. Be not misled by those who teach that we must be realistic and tough—tough in business, tough in bargaining, tough in negotiation. What we really need is toughness in spirit; that toughness that makes us cling to our ideals though the whole world seem against us.

And if by precept and example you can teach your children to cherish their ideals, to let the happiness come out of their hearts, which is its natural home; if you can teach them that joy and satisfaction are to be found in the spirit of giving, in their daily work, in tolerance and understanding, in humility and in faith, then you will have done them a double service, for you will have put their feet upon the path that leads to a happy and contented life, and you will have shown them how to leave the world a little better than they found it. In short, you will have taught them the secret of living.

As one who breaks the rules so sadly, I put these things before you in all humility.

SUDECK'S ATROPHY AND CORTISONE.

By A. F. DWYER,
Sydney.

THERE have been many excellent descriptions of the clinical aspects of Sudeck's atrophy. However, it is an uncommon condition in its most fully developed form, and for that reason some misconceptions exist in regard to it.

In a recent issue of THE MEDICAL JOURNAL OF AUSTRALIA the reviewer of Key and Conwell's book "The Management of Fractures, Dislocations and Sprains" made the following comment:

> In the chapter on complications, so-called Sudeck's disease is described as a curious complaint that has never been produced experimentally; but the common view now is that it is more likely to be a grand example of successful malingering, or the effects of disuse atrophy in an honest but timid and elderly patient. The late result of prolonged immobilizations of the hand or foot, whether associated with the dishonest purpose of making a claim for permanent disability, or from any other cause, is the same—namely, stiffness of the fingers and adhesions in the tendon sheaths. These changes may be permanent if the pretence has been kept up too long; but they often clear up reasonably soon after a lump sum settlement.

In the past nine years only two cases of Sudeck's atrophy, in the classical sense of the term, have been treated at the fracture clinic of Saint Vincent's Hospital, Sydney. The first patient was seen in 1943. He was a well-built man of thirty-eight years, who developed the condition as a sequel to a minor wrist injury, without fracture. He came to the fracture clinic six weeks after injury and the typical changes were well established. The second patient was an active, intelligent woman of fifty-two years, who was treated last year and whose history will be given later. Neither of these patients was entitled to compensation. They were not timid, nor could they be called elderly.

During the same nine years well over 10,000 new patients would have been treated, and amongst them many examples of minor degrees of post-traumatic reflex dystrophy were found. A malingerer may well mimic a minor dystrophy, but no man could consciously produce the severe degree of fibrosis which is seen as early as six to eight weeks after injury in a true Sudeck's atrophy.

It may be true that "Sudeck's disease is a curious complaint that has never been produced experimentally". However, the "formalin arthritis" produced by Hans Selye appears to resemble Sudeck's atrophy much more closely than it does rheumatoid arthritis.

Clinical Features.

The condition is most commonly seen after minor injuries in the region of the wrist joint, but it occurs in the lower limb as well. A typical story would be as follows.

A man sustains a fracture or a contusion in the region of the wrist. On careful questioning he appears to have much more pain than the objective nature of the injury would lead one to expect. Over the next few days, in spite of good immobilization, the pain does not abate, but spreads to involve the entire hand and fingers.

Within a week it is noted that the fingers are puffy and that movement, originally good, is now poor. The patient states that it hurts him to use the hand, and will usually say that he feels a continual burning pain throughout the hand and that this is especially evident at night. Untreated, the pain extends in a glove and stocking manner up the limb and "trophic" changes occur in the hand. These nutritional changes affect the skin and its appendages, the joints, tendon sheaths, palmar fascia, and bones.

The skin is warm, usually dry and somewhat red and shiny. There is a deficiency of hair on the backs of the fingers; the nails grow more rapidly; they are striated and curved, and raise subungual pads; the fingers show conical atrophy; there is a fusiform, tender swelling of all small joints and there is palpable thickening with early contracture of the flexor sheaths and palmar fascia. At this stage the skiagrams reveal a spotty decalcification of all bones, most marked in the cancellous regions, and a suggestion of narrowing of the "joint spaces"—thinning of articular cartilage.

All these changes are well in evidence at six weeks and by this time the patient has only a few degrees of movement at the metacarpo-phalangeal and interphalangeal joints, and varying degrees of restriction of movement at all other joints of the limb. The shoulder is especially liable to become stiff.

Ultimately the pain generally subsides after many months and the patient is left with a hand extensively infiltrated with fibrous tissue and permanently crippled to a varying degree. However, De Takats states that cases have been known in which the glove-and-stocking spread of pain has gone up one limb and affected the other limb and trunk, and the patient has been ultimately confined to a mental asylum or has committed suicide.

Ætiology.

The organic changes are fundamentally based on a generalized vasodilatation and increase in capillary permeability. This permits all tissues to be soaked in a protein-rich exudate which initially causes a readily reversible stiffness of joint capsules and tendon sheaths, but soon fibroplasia occurs, adhesions form, and gradually permanent contractures develop.

The underlying neuro-vascular mechanism is related to the abnormal state of affairs seen in causalgia, various minor nerve irritations, *osteitis pubis*, and even *herpes zoster*, in which irritation of a nerve root causes hyperæmia and vesicle formation in the skin.

The experimental prototype of all these reactions is the triple response of Lewis (see Figure I). Injury at point A causes liberation of H substance from damaged cells of the skin. This leads to local arteriolar and capillary dilatation and increase in capillary permeability. In addition, by means of an axon reflex, arteriolar and capillary dilatation takes place at point B, and in this manner a flare occurs about the point of injury. Lewis suggested that the axon reflex was mediated by the liberation of some chemical substance, possibly histamine. Others have suggested acetylcholine as a chemical transmitter.

The triple response is a normal sequel to any injury or infection and its obvious purpose is to mobilize antibodies, phagocytes and the necessary metabolites at and about the point of damage.

The experimental investigations of Lewis on cutaneous hyperalgesia are of particular interest in connexion with Sudeck's atrophy. He found that various forms of injury to the skin—electrical, mechanical, and thermal—could give rise to a long-lasting state of hyperalgesia, which involved a considerable surface area surrounding the point of injury. The reaction lasted for different periods in different individuals, and according to its original intensity. In full reactions it was usually recognizable eight to twelve hours subsequently. He also noted that with full soreness a sense of very slight spontaneous burning or smarting was felt.

He was able to show quite conclusively that the reaction was brought about by axon reflexes causing the liberation of a chemical substance in the skin. This chemical substance resulted in the unpleasant sensations and also vasodilatation, and he regarded the whole reaction as a normal defensive mechanism.

We thus have two normal physiological neuro-vascular responses to injury—an early triple response and a long-lasting and widespread hyperalgesia. Almost certainly Sudeck's atrophy is a perversion of these normal mechanisms, but details are a matter of conjecture.

A useful working hypothesis is as shown in Figure II. Injury at point A results in the liberation of a histamine-like substance at point B. This liberation is abundant enough to cause not only vasodilatation and an increase in

capillary permeability at point B, but also to engender an axon reflex affecting point C. From C the vascular changes spread by a further axon reflex to point D *et cetera*.

This is the bare bones of an hypothesis, and no attempt has been made to discuss Lewis's postulation of a "nocifensor nervous system", the arborizations and overlappings of the axons of pain fibres, or the place of spinal reflexes. However, it has the virtue of explaining how widespread effects can evolve from a local injury and how various therapeutic measures may meet with success.

Studies in hyperalgesia are also relevant to the fact that injuries are common, but severe neuro-vascular dystrophies are rare. Lewis found great variability in the ease with which hyperalgesia could be induced, and noted

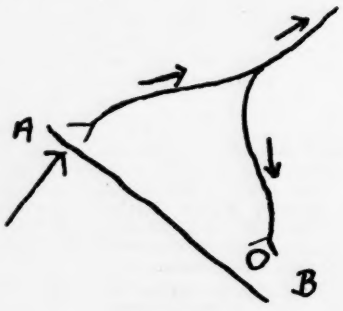

FIGURE I.

that it did not develop in certain individuals. He also had good grounds for suspecting that the mechanism could be put into action by the central nervous system.

We may note in Sudeck's atrophy that:

1. The initial injury is usually associated with severe pain. Anyone who has suffered from a subungual hæmatoma knows that an injury does not need to be gross in order to be painful. Many direct injuries in the neighbourhood of joints do cause severe pain.

2. There is a tendency for the condition to develop in more highly strung individuals and in people who have

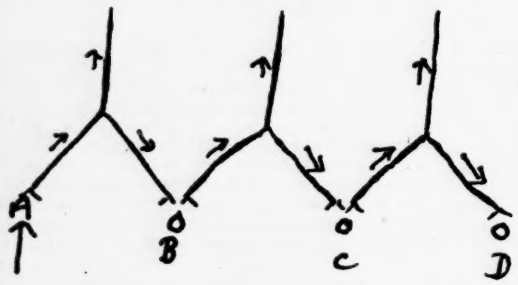

FIGURE II.

some worry or other. Anxiety about the medico-legal aspects of an injury or its possible effect on a man's future earning capacity may well intensify any local neurovascular reaction.

Treatment.

Any measure which increases blood flow through the extremity will benefit patients suffering from Sudeck's atrophy. If the rate of perfusion of the tissues is sufficiently great, "H substance" is washed away rapidly as it forms and the vicious cycle of axon reflexes is broken.

Exercise is the underlying basis of all treatment. It does much more than just stimulate blood flow through the limb. Exercise combats adhesion formation, improves the nutrition of synovial and joint tissues, and maintains muscle tone. No matter what other measures are taken, the patient must engage in active exercises of all muscle groups throughout the course of the disease.

It is possible for a resolute patient, suitably encouraged, to cure himself by exercises alone, and in fact the first patient mentioned did effect a cure in about four weeks, by exercise. Active use of the limb, carried out either spontaneously by the patient or at the insistence of the attending surgeon, has undoubtedly prevented the development of "trophic changes".

Sympathetic Surgery.—Sympathetic block by "Novocain" and sympathectomy is the most effective means of causing vasodilatation we possess, and "Novocain" infiltration, repeated as often as necessary, should be carried out as soon as a diagnosis of severe reflex dystrophy has been made.

Case Reports.

CASE I.—V.G., a widow, aged fifty-two years, was accidentally struck on the ulnar border of her right wrist at a party on March 17, 1951. The blow caused her very severe pain at the site of injury and the whole forearm and hand were aching when she came to the casualty department of Saint Vincent's Hospital half an hour later. An X-ray examination showed an oblique fracture of the distal part of the ulna without displacement. The arm was splinted and she was told to attend the fracture clinic. She was first seen at the clinic on March 19, 1951, and at varying periods of two to five days thereafter.

When I questioned the patient about six weeks later she told me that the pain was initially a severe ache which worried her continually, but within a few days her chief complaint was of a superficial "burning pain" of the hand and fingers, especially obvious at night.

The fracture registrar initially noted that finger movements were satisfactory. It was not until four weeks after injury that he saw any need to worry about these—they had depreciated considerably since she was originally seen and, moreover, considerable swelling of the forearm and hand was present.

The patient's burning pain continued and movement range continued to lessen. Five weeks after injury the objective state of the hand was such that the registrar made a wrong, but understandable, diagnosis of low-grade cellulitis of the hand. The hand was swollen, œdematous and warm, and finger movements were very restricted—passive extension and flexion of the fingers not only appeared to be organically limited, but caused pain radiating up the arm. When the condition failed to respond to chemotherapy, he asked me to see the patient.

By this time six weeks had elapsed and all the trophic changes were well evident—warm, red, glossy skin, with striated nails, pulp atrophy, subungual pads, joint swelling and thickening, and contracture of the flexor sheaths and palmar fascia. An X-ray examination showed the healing fracture and very pronounced spotty atrophy of all bones. The atrophy of the bones was far greater than is seen in a usual disuse atrophy after six weeks.

The fingers were held in a resting position of partial extension—the position adopted when waiting to catch a ball—with flexion gradually increasing from fore to little finger. From this attitude the patient could actively extend the fingers only very slightly, and flex them not much better, the total excursion of the finger tips being about an inch.

In addition, she complained of pain and stiffness of her shoulder and was unable to raise her right hand above shoulder level. On examination all gleno-humeral movements were restricted, but especially abduction, external and internal rotation, as is the case with most stiff shoulders.

She was immediately referred to the neurosurgical unit for sympathetic block, and instructed to attend the physiotherapy department daily. The importance of exercise was carefully explained. She gained immediate relief of pain from the sympathetic block, but her movement range showed no dramatic improvement. The neurosurgeon did not feel that repeated blocks would be worth while, and so it was decided to concentrate on exercise. The patient was kept under continued observation, but made practically no gain in movement of the fingers or the shoulder, and the trophic changes did not improve.

It was felt on theoretical grounds that cortisone should be of benefit. However, the drug was expensive and in short supply, and as no one appeared to have any experience with its use in this complaint, the patient was not admitted to hospital until September 4, 1951, nearly six months after the onset of the dystrophy. She was given 300 milligrammes of cortisone the first day, 200 milligrammes the second day, 100 milligrammes the third day and daily thereafter until a total of 1·0 gramme had been used. In order to avoid a possible letdown, she was then given 30 units of ACTH for two days. The total period of treatment occupied ten days and no side effects attributable to the drug action were noted.

Within twenty-four hours of the commencement of cortisone all shoulder pain had disappeared and for the first time in months the patient could reach above the shoulder level. Within three days all pain, and much of the subjective feeling of stiffness, had left the hand and an obvious gain in movement range was apparent. At the end of treatment the hand still showed some spindle swelling of the joints, the palmar fascia and tendon sheaths were still thickened and contracted, and only a slight gain in extension had been made. However, flexion was greatly improved and she could close all her finger tips to within three-quarters of an inch of the palm. At the same time the range of shoulder movement had increased to give her complete restoration of function at this joint.

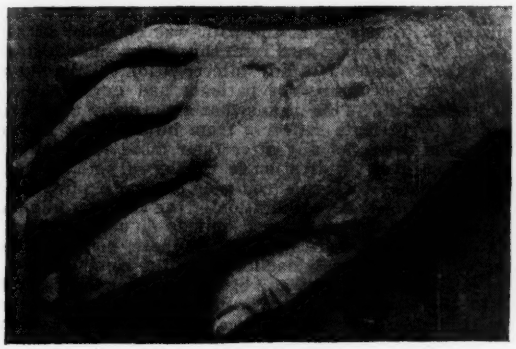

FIGURE III.

Immediately on leaving hospital the patient returned to work. She showed no tendency whatever to relapse and has been able to use her hand quite satisfactorily. Six months have now elapsed and some slow improvement appears still to be taking place in the general state of the limb. However, she still cannot fully extend the fingers and still cannot actively close the fingers into the palm.

I regard this patient's residual disability to be due to well-established fibrosis of her joint capsules, tendon sheaths and palmar fascia, and strongly doubt if the hand will ever be completely normal.

CASE II.—Mrs. I.B., aged sixty-five years, was admitted to Saint Vincent's Hospital under the care of Mr. W. Hugh-Smith. The patient was initially cared for in the country. She had suffered a fracture of the lower end of her right radius, which was reduced and immobilized for four weeks. Apparently the fingers were swollen and painful from the time of the fracture, and her range of movement failed to improve after immobilization was discontinued. About two months after the injury she had also begun to notice stiffness and pain of her right shoulder.

She was admitted to hospital on December 21, 1951, three months after the injury, and at this time was complaining of a constant ache of the hand and a subjective sensation of stiffness, and said that she could not open or close her fingers properly. Her fingers were swollen and puffy and the range of movement can be seen from the photographs (Figures III and IV). However, she did not show the strongly marked spindle swelling of the interphalangeal joints or the palmar fibrosis of the first patient.

She complained also of pain and stiffness of the shoulder and showed a generalized limitation of gleno-humeral movement to a degree where she could merely raise her hand sufficiently to touch her ear and could not put it behind her back.

Cortisone was commenced on December 21, 1951. She received 300 milligrammes on the first day, 200 milligrammes on the second day, 100 milligrammes for five days, 50 milligrammes for nine days, and 25 milligrammes for two days. She was then given ACTH, 30 units the first day, 30 units the second day, 20 units the third day, and 10 units for four days.

She was kept in hospital for twenty-seven days and improved continually, so that on discharge she had a completely normal hand and almost full shoulder movement. The relatively long hospitalization was partly due to the fact that she was a country patient. Also it is probable that more cortisone and ACTH were given than was strictly necessary, but no side effects were noted.

When seen again at an Orthopædic Association meeting six weeks later, her hand was still normal and she had full use of the shoulder, but on examination a trace of limitation of external rotation and abduction persisted.

I do not regard this patient as having suffered a full-blown Sudeck's atrophy. Three months had elapsed between injury and attending Saint Vincent's Hospital, and no relentless fibrosis was evident. She had never suffered burning pain and did not have red, shiny, sensitive skin. However, her skiagrams revealed well-marked spotty atrophy of the bones of the limb up to the shoulder, and I would regard her as having suffered a moderately severe post-traumatic dystrophy.

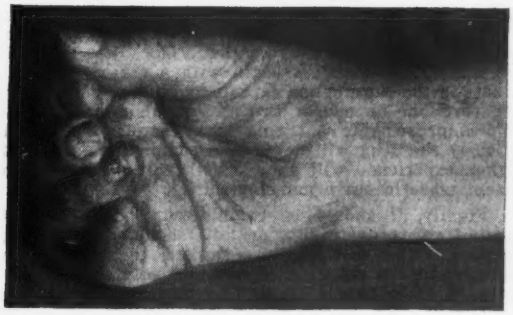

FIGURE IV.

Discussion.

1. It is probable that Sudeck's atrophy is merely a post-traumatic reflex dystrophy in its most severe degree, but in my experience it constitutes a very clear-cut clinical condition. When a patient complains of continual burning pain superficially situated in the skin, when the skin is shiny, warm, and irritable to touch, then we may expect rapid development of joint fibrosis and fascial contracture. The minor dystrophies are quite common and have a considerable nuisance value, but the patient never complains of more than a severe ache and the condition will generally respond to sufficiently persistent exercises. A similar clinical distinction is evident between a true causalgia as described by Weir Mitchell and the various minor nerve irritations, which frequently follow nerve injuries in the upper limb.

2. In the two cases reported cortisone appears to have had a dramatically beneficial clinical effect. Three possible reasons for its action come immediately to mind. The first is psychological. This is possible, because emotions can influence the peripheral circulation, but quite unlikely. The second possible reason has to do with the inhibitory effect of cortisone on fibrous tissue formation. This is unlikely, because the first patient had fibrous tissue formation in evidence before treatment was begun and still had the fibrous tissue at the end of treatment. Possibly very prolonged cortisone administration would have improved the diffuse fibrosis, but we did not feel justified in using so much of a scarce drug to find out. The third possible reason is that there is a direct action on the capillary circulation. The water-logging of all tissues with protein-

rich exudate is the initial cause of stiffness and remains an additional cause of stiffness for a long time after fibrosis begins. Cortisone apparently antagonizes the liberation of some histamine-like substance, judging from its effect on *status asthmaticus* and other allergic conditions. It is very likely that a similar effect was responsible for its beneficial action on these patients.

3. The permanent effect of treatment is to be expected. Sudeck's atrophy is a self-perpetuating vicious cycle condition, probably sustained by liberation of a histamine-like substance in abnormal amounts. It persists long after the initiating lesion has healed, and once the cycle is broken by cortisone or any other means, there is no tendency for it to recur. In rheumatoid arthritis and many other complaints in which cortisone is of benefit, the basic cause remains untouched and relapses are depressingly regular.

4. The concomitant cure of the stiff shoulders is of interest. "Frozen shoulders" are common complications of injuries to the shoulder itself, of fractures in the upper limb and of brachial neuritis, due to cervical nerve root compression. Treatment of brachial neuritis by neck traction is sometimes accompanied by cure of the stiff shoulder, and I have seen stiff shoulders respond on a couple of occasions to a sympathetic block. It is possible that many stiff shoulders begin with a reflexly generated exudate in the capsule and capsulo-tendinous cuff. We have tried cortisone on one or two stiff shoulder patients with rapid improvement; but this is a complex subject and obviously the drug will never be a complete answer. Only further experience will show whether it deserves a permanent place in our armamentarium.

5. Finally, it should be emphasized that in very few cases of reflex dystrophy, or stiff shoulder, is cortisone indicated. The vast majority respond to active exercise instituted at the proper time and correctly supervised. A few need to be helped along by sympathetic block. In a recalcitrant case it would always be worth while using cortisone before resorting to sympathectomy, but the decision to use cortisone in true Sudeck's atrophy must not be too long delayed. It must be remembered that fibrosis occurs rapidly and resolves slowly and imperfectly, and is probably little influenced by the action of the drug.

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SOME ASPECTS OF CHOLECYSTECTOMY.

By J. K. MOWAT,
Brisbane.

THE case histories of 400 patients subjected to cholecystectomy at the Brisbane General Hospital during the period June, 1949, to June, 1951, have been reviewed and analysed in some detail.

It is well known that statistical surveys bristle with almost insuperable difficulties in the matter of their accurate interpretation, and that moderately large numbers by no means overcome these difficulties. Nevertheless, much can be learnt by a panoramic view in retrospect of work that has been done, and the findings in this investigation are thought to be not entirely uninstructive.

The total number of cases of cholecystectomy, or common bile duct exploration, or the two combined, was 400.

1. The symptoms complained of in relation to abnormality found were as follows: (i) Typical severe biliary colic, 40%; of these, (*a*) small stones were present in 92%, (*b*) large stones only in 5% and (*c*) no stones at all in 3%. (ii) Moderate pain of unequivocal biliary type, 34%; of these, (*a*) small stones were present in 70%, (*b*) large stones only in 15% and (*c*) no stones at all in 15%. (iii) Dull aches, constant pains, flatulence *et cetera*, 21%; of these, (*a*) small stones were present in 60%, (*b*) large stones only in 30% and (*c*) no stones at all in 10%.

I am inclined to regard the last group, (iii), as consisting of functional dyspepsia, abdominal neurosis, visceroptosis and the like, in which "silent" stones are revealed by radiography. It would be interesting to ascertain the relief rate as a result of operation, and an effort may be made to pursue this group with that object in view. It is my own opinion that in very many instances these patients are not benefited symptomatically by operation. From group (i) it is apparent that typical severe biliary colic, in which the patient rolls about and describes the pain as agonizing, is in nearly all instances due to small stones, of which one has entered the cystic duct.

2. An unequivocal history of passing very dark urine following some attacks of the pain was obtained in 30%. Doubtful cases were omitted from this figure, some histories were without mention of any interrogation on the subject of urine, and a number of patients had never observed their urine at all. This means that abnormally dark urine is passed by patients suffering from biliary colic in probably 50% of cases, usually after their more severe attacks of pain. Such an observation is worthy of note, and it is to be concluded that this common clinical finding is no indication in itself for exploration of the common bile duct.

3. Jaundice reliably observed outside or actually seen in the hospital was present in 17%. Of these, no stones were found either in the gall-bladder or in the common duct in three cases, although the gall-bladder was undoubtedly the seat of the underlying disease process. In cases such as these it is clear that the jaundice must be due to calculous obstruction of the common duct by a stone which the surgeon fails to discover or which has passed into the duodenum prior to operation or else to partial obstruction by œdema.

4. The incidence of common duct exploration was 77 cases (that is, 20%). The chief apparent reasons for exploration were as follows: (*a*) recent jaundice, 48%; (*b*) past jaundice, 12%; (*c*) dilated common duct, 30%; (*d*) stone palpable in the duct, 5%; (*e*) the presence of small stones in the gall-bladder together with a dilated cystic duct, 25%. It is to be noted that the indications for duct exploration vary widely from surgeon to surgeon and that there are many personal proclivities and idiosyncrasies. Undoubtedly the actual technical difficulty or otherwise of assaulting this structure is a deciding factor in not a few cases, when the indications are equivocal. However, the overall incidence of exploration is more or less equivalent to the figures from other clinics, as is the figure below for the incidence of stones found in the common duct.

5. Stones were removed from the common bile duct in 31 cases—that is, from 40% of the ducts examined. Nearly all of these choledochostomies were closed around a short "T" tube which did not enter the duodenum, and in about three-quarters of the cases cholangiography was performed before the tube was withdrawn. This is in contradistinction to the practice in some American surgical units (for example, Cattell, 1948), where a long distal limb of the "T" tube protrudes through the duodenal papilla, so that cholangiography can give no information.

6. The duct was not explored despite recent jaundice in 30 cases. These are difficult of analysis and dissection, because the operative details are written by the resident surgeon acting as assistant, and the reasons for negative decisions are rarely entered. However, one can speculate on a number of reasons for avoiding choledochostomy even in the presence of this very well-known indication, the chief of which is probably the finding of a duct normal to inspection and palpation together with a narrow cystic duct and at the same time some restriction in the exposure

of the field. It is noteworthy that these 30 patients, almost without exception, proceeded to normal recovery; but here again an adequate follow-up would be highly instructive. A few at least would be expected to be afflicted later with common duct obstruction.

7. The incidence of mucocele was 3%. The mucoceles were all typically large and thin-walled, and in all a stone was firmly fixed in the cystic duct.

8. The incidence of empyema was 2%. Note that cases of cholecystostomy are not included in this survey, so that this figure is not an indication of the frequency of empyema of the gall-bladder.

9. The incidence of carcinoma of the gall-bladder was nil. However, this disease is by no means rare, and in the opinion of some surgeons it is a reason for prophylactic cholecystectomy in cases of silent calculus disease. A steady flow of occasional cases is seen at this hospital.

10. The incidence of acute cholecystitis found unexpectedly at the operation of routine cholecystectomy was 10%. Œdema of the gall-bladder wall was the sole criterion used in selecting this group, and it was taken from those cases diagnosed clinically as of uncomplicated biliary colic—that is, in which pyrexia and peritonism were absent. In no instance was the œdema such as to preclude the safe performance of cholecystectomy, but nevertheless this group is not without some academic interest.

11. Previous cholecystostomy had been performed in seven cases—that is, 2%. This group is worthy of note and calls for further study over a large number of cases. To ascertain the incidence of cure following this operation one would need to pursue 50 or 100 patients over a ten-year period subsequent to the drainage. The figure given here, of course, bears no relation at all to the recurrence rate of calculus disease after cholecystostomy. Pye (1951) is of the opinion that this is a good operation and is performed too infrequently, for it has been his observation over a period of twenty years at the Brisbane General Hospital that surprisingly few of these patients return with further serious trouble.

12. Choledochotomy was performed in 14 instances; that is, the common duct was opened, explored and closed with drainage of the peritoneal cavity only. These patients all did well and this operation is considered in Brisbane to be safe.

13. Exploration of the common bile duct following previous cholecystectomy was performed in eight cases. Of these, stones were found in five cases and no stones were found in three cases. One might comment here upon the well-known elusiveness of calculi, even large ones, in the common and hepatic ducts, and upon the extraordinary difficulty, or even rather the impossibility, of being certain that all stones are removed therefrom at the conclusion of operation. The ether irrigation method of Pribram was used in a few instances in this series, apparently with satisfactory results, in that cholangiograms became normal following the treatment. Patients do not like having ether in their bile passages.

14. The post-operative mortality was as follows: cholecystectomy, 0·5%; cholecystectomy with exploration of the common duct, 5·0%. These two figures are a little lower than one expected to find when it is borne in mind that in a large public hospital there is a steady trickle of serious and desperate cases. However, resuscitation teams with their aspiration tubes and their rituals of intravenous replacement have salvaged large numbers of patients who surely otherwise would have been lost.

Although these results are reasonably good, they are drawn from the wards of trained surgeons whose experience is supplemented by well-organized ancillary services. It is my opinion that in general practice the dangers of cholecystectomy are underestimated by most occasional surgeons, and that unpropitious results are put down to bad luck with undue readiness.

Indications for Operation on the Biliary Tract.

It is proposed here to pass over a dozen or so more or less uncommon conditions such as pancreatic carcinoma, pressure from malignant nodes, neoplastic stricture, hydatid colic, newgrowth of the gall-bladder and the like. More than 90% of operations are performed for the relief of the adverse effects of gall-stones or gall-bladder inflammation, so that in general the indications for surgical interference may be enumerated as hereunder.

In Cases of Gall-stones.

The History and Symptom Complex.

The pattern of the pain is important. Recurrent attacks of violent right upper abdominal pain which cause the patient to wriggle about, to cry out, and to vomit, are typical of gall-stone colic. If some of the attacks are abrupt in onset, if some relent suddenly and completely, and if there is radiation to the right shoulder blade, then operation may be advised on the ground that the story itself is pathognomonic.

But the pain may be less severe than this, be centrally placed high in the epigastrium, and exhibit no radiation. It may have a varying relationship to the consumption of food or to certain particular foodstuffs. Or there may be an element which moves to the left, or down to the umbilicus, or to the right lower quadrant. The pain, on careful interrogation, may turn out to be a dull ache at the right costal margin, more or less constant and with exacerbations from time to time, usually after meals.

Under any of these circumstances further confirmation of the diagnosis is required, as outlined in subsequent sections.

The history of having passed very dark urine, like strong tea, after an attack of pain is important. Provided that this information is unequivocal, it is excellent evidence that biliary tract disease requiring operation is present.

The history of having passed clay-coloured stools is less often obtained, requires corroboration, and of course is indicative of non-entry of bile into the duodenum over a period of twenty-four hours or more, owing in most instances to common duct obstruction by a stone.

The history of jaundice must be carefully confirmed and a description of its onset, duration and depth obtained. Many lay persons describe a sallow look as jaundice. It is generally held that a history of jaundice is an indication for exploration of the common bile duct.

The complaint of flatulence may be made. Many people suffer from "the wind, doctor", and sensations of fullness and epigastric discomfort are so common as to be valueless as a symptom of any specific organic disorder. However, flatulent dyspepsia is a well-known feature of gall-bladder disease.

An intolerance of fats is an equally common finding and is of significance.

The absence of symptoms referable to other organs, notably the heart and right kidney, is important.

The Physical Examination.

If an attack of biliary colic is witnessed it can be recognized as such nearly always, although it is wise not to be precipitate or glib in this decision, but to give passing thought to the coronary vessels and the pelvis of the right kidney. If there are tenderness and guarding in the right upper quadrant, with some degree of pyrexia, it may reasonably be concluded that acute cholecystitis is present and due to cystic duct obstruction by a stone.

In interval cases examination of the patient may give entirely negative results. However, more often there is some abnormal finding such as tenderness on deep palpation and the eliciting of Murphy's sign. The gall-bladder may be palpable as a tense globular swelling projecting from beneath the ninth costal cartilage towards the umbilicus, smooth, descending with inspiration, and dull to percussion. An equally definite mass is sometimes palpated and found at operation to comprise omentum and transverse colon in an adhesive conglomeration around the diseased gall-bladder. Provided one can exclude a Riedel's lobe of the liver and a palpable normal right kidney, any mass is, of course, an indication for operation; deep tenderness and the presence of Murphy's sign are not—that is,

without good confirmation in the history and in the special investigations.

The Ancillary Investigations.

Plain X-Ray Examination.

Various discoveries of moment may be made as a result of plain X-ray examination, a simple and more or less inexpensive procedure. These are as follows: (*a*) A cluster of faceted calculi may be seen in a position corresponding with that of the gall-bladder. Provided the history has a biliary flavour, these are undoubtedly the cause of the symptoms and should be removed. (*b*) A solitary rounded shadow cephalad to the renal outline is probably a cholesterol *solitaire*. Confirmation will often be required by intravenous pyelography or cholecystography, or both in combination. The single large stone does not give rise to typical biliary colic and is often symptomless. Careful correlation of the symptomatology with the physical findings is required here, as outlined above, in order to be reasonably sure that cholecystectomy will bring about relief from the troubles complained of. In most cases operation will be indicated, and in this regard one may bear in mind that prophylactic surgery is not unreasonable, because acute cholecystitis occurs quite frequently as a result of cystic duct obstruction by a solitary stone of large size. (*c*) In the undiagnosed acute abdominal emergency an absence of air beneath the diaphragmatic dome will help to exclude a ruptured ulcer, and absence of dilated coils to exclude intestinal obstruction and acute pancreatitis (Grollman, Goodman and Fine, 1950). The indications for operation in acute cholecystitis are discussed below.

Cholecystography.

Maitland has recently (1952) discussed cholecystography as a method of investigation at some length. It would appear that the more care taken in the production and study of serial films following the fatty meal, the more often will disease be demonstrated radiologically. In America great reliance is placed upon cholecystography (for example, McKell, Lahey Clinic, 1948), whereas in this country most experienced surgeons look upon the test as a valuable but subsidiary adjunct to careful clinical survey. In the Brisbane General Hospital it is the practice to make extensive use of Graham's test, and the divergence between operative and radiological findings is sometimes rather startling. It is my own view that cholecystography is unnecessary in not a few instances of typical biliary disease.

Let us see what conclusions are to be drawn from a few typical radiological reports.

(*a*) "The gall-bladder fills with a good concentration of the dye and empties normally in response to a fatty meal. No calculi are visible." Does this mean that the organ is normal? Certainly not. Many a viscus replete with several families of faceted calculi has been removed after just such an X-ray report. The history of the symptoms and the pattern of the pain must in all cases override the evidence of a normal response to Graham's test. However, the test may well be repeated and the phase of emptying perhaps more painstakingly observed.

(*b*) "The gall-bladder does not fill." Does this mean that operation is advisable? Not at all. One's object in subjecting any patient to operation is either to cure his symptoms or to prevent the development of serious disease. Prophylactic gall-bladder surgery, surely, is seldom warranted, so that once again one must have recourse to an analysis of symptoms. If these are vague, consisting mainly of flatulent dyspepsia, full feeling and eructations, with occasional sessions of pain of the dull ache or dragging type, they are likely to continue more or less unchanged after cholecystectomy. This is equally true if Murphy's sign is present, but not if the gall-bladder is palpable. Patients such as these are best treated medically by diet and chologogues and observed over a prolonged period before operation is advised. The subject of the medical management of biliary disease is dealt with by Jordan (1948) in a way that will repay perusal. (*c*) Calculi are visible, often as negative shadows in the dye. This type of finding is similar to the corresponding findings on plain X-ray examination and is dealt with above.

Excretion Pyelography.

Excretion pyelography will exclude most cases of gross renal disease, such as hydronephrosis and calculus disease.

Electrocardiography.

Electrocardiographic investigation is a wise precaution in cases that are other than typical. The coexistence of quiet coronary occlusion and cholelithiasis has been seen more than seldom. In one notable instance in my own practice a classical picture of gall-stone colic altered in the third attack so that an element of the pain radiated down the medial aspects of both arms. Electrocardiographic examination after several days revealed unequivocal evidence of myocardial infarction, and cholecystography revealed many faceted calculi which were later removed. He has had no further attacks in four years.

Barium Meal Examination.

Barium meal examination has an obvious place in these investigations and need not be much enlarged upon, except to mention that diaphragmatic hernia is a great simulator in the upper abdominal syndrome and should be borne in mind by all radiologists, whether it is queried by the clinician or not. McKell (1948) states that it is routine practice at the Lahey Clinic to investigate the stomach and duodenum radiologically prior to performing cholecystectomy, on the grounds that a gastric or duodenal lesion is potentially more serious than a biliary lesion, and in the absence of this precaution silent stones will occasionally be removed, the real cause of the patient's trouble being left *in situ*. This sounds not unreasonable. However, I imagine there are few surgeons in Australia quite so cautious as to proceed in this way when reasonably satisfied with their diagnosis. On the other hand, it may be said here in parenthesis that a failure to examine thoroughly at operation all the adjacent organs, including the kidneys shows an incautious and over-confident attitude which is not in the best interests of the patient.

Other Investigations.

A serum bilirubin content greater than 0·3 milligramme per 100 millilitres means that subclinical jaundice is present.

Serum amylase and lipase and urinary diastase estimations are of value in some cases when there is a thought that pancreatic disease may be the cause of symptoms. If these are significantly raised twelve to twenty-four hours after an attack of pain, then it is probable that cholecystectomy is still indicated, but perhaps in conjunction with other procedures designed to relieve the state of recurrent pancreatic œdema (Mowat, 1951).

The Casoni reaction and a selection of the numerous liver function tests will be used on occasion.

In Acute Cholecystitis.

Although varying ætiological factors are occasionally concerned, acute cholecystitis is probably best regarded as a complication of gall-stones and as due to obstruction of the cystic duct. Its diagnosis is usually not difficult and has been dealt with previously. However, what is to be done about it? There is a tendency in the United States of America to compare this ailment with acute appendicitis and to ablate the offending organ without any ado. Statistical figures on the frequency of perforation and gangrenous cholecystitis are produced as evidence of the dangers that must attend expectant treatment (Thomas and Womack, 1949).

It always seems to me odd that a surgeon can formulate a set policy and routine method of conduct in a disease that varies from case to case in its severity and in the stage at which it is first seen. We are called upon to treat the patient, surely, and to relieve him of his affliction, not to regard him as a sort of unfortunate encumbrance temporarily obscuring his diagnosis. In other words, let there

be no rule of thumb in this matter. However, the following points may be borne in mind: (*a*) In most cases acute cholecystitis will subside on treatment by starvation, rest and gentle sedation. (*b*) If the patient is first examined forty-eight hours or so after the onset of symptoms, cholecystectomy is likely to present technical difficulties owing to gross œdema and infiltration of the tissues. (*c*) In early cases immediate cholecystectomy has much to recommend it, especially in the earlier age groups. The overall period of disability is shortened. (*d*) The indications of impending perforation are fairly clear-cut and amount in effect to a general worsening of the clinical picture. A persistence of vomiting is important if associated with any spread at all in the area of tenderness or increase in the degree of peritonism. The pulse rate rises early. It is found at the Brisbane General Hospital that senior surgical registrars can recognize impending perforation with accuracy and confidence. (*e*) Cholecystostomy is the operation of choice in most cases in which conservative measures have been insufficient. An effort is made to extract the stones from the cystic duct. (*f*) When the acute condition subsides, as it does in the majority of instances, interval cholecystectomy is to be advised and performed in six to twelve weeks—preferably the latter, as œdema and thickening of the tissues remain for a surprisingly long time. (*g*) Acute cholecystitis differs basically from acute appendicitis, in that cultures from early cases do not produce a growth of virulent pathogens.

This knowledge is essential to an understanding of the fundamental nature of the disease and to the development of a balanced outlook on the subject of its treatment. It is also well to bear in mind that an empyema of the gall-bladder does not contain pus but only a material that looks like pus; it is a mixture of calcium salts, cholesterol and mucus, and although admittedly infectious, it is not loaded with the death-dealing bacteria that are present in an appendiceal abscess.

In Empyema of the Gall-bladder.

Empyema of the gall-bladder is best regarded as a stage in the late development of some examples of acute cholecystitis, in which the calculous obstruction to the cystic duct is complete and unrelenting and the degree of virulence of secondarily infecting organisms is not especially high. The patient with acute cholecystitis fails to continue to improve, he has persistent pain of moderate severity and considerable anorexia. A tender mass may be palpable as the guarding subsides.

Cholecystectomy is the operation of choice in empyema. Nothing much is likely to be gained by expectant delay. However, major technical difficulties may be encountered in displaying the cystic duct and artery in the presence of adhesions and tissue œdema, and under these conditions there should be no hesitation in reverting to cholecystostomy with removal of the obstructing stone if it can be dislodged. One may here emphasize the more or less obvious fact that if an obstructing calculus is left in the cystic duct at cholecystostomy, the patient will be relieved of his immediate troubles but is likely to be left with a permanent fistula discharging mucus, which will call for a further operation. It may be said that further operation is necessary anyway, because cholecystostomy is unsatisfactory; but this is not so, especially in the cases of patients of advancing years, many of whom suffer from no more symptoms for the rest of their days after this conservative surgery.

In Mucocele.

In mucocele there is usually a history of recurrent attacks of pain similar to biliary colic which give way to a constant dull ache in the right upper quadrant of the abdomen, with flatulent dyspepsia. Complete obstruction to the cystic duct has occurred as a result of a calculus lodging firmly in its lumen (or, of course, to a neoplasm at the junction of the ducts; but this is rare and is not under discussion here). The gall-bladder is not inflamed and is fairly thin-walled and distensible, so that it is likely to be readily palpable through the abdominal wall.

The diagnosis of mucocele is as a rule not especially difficult provided the story is in keeping with that of biliary disease; but in some instances the symptoms are vague, and in others the palpable mass lies lower down in the mid-abdomen, so that other conditions, notably some renal abnormality, are to be excluded. In mucocele the mass is not palpable bimanually with one hand in the renal angle (that is, it is not ballottable) as are most retroperitoneal masses, and the cholecystogram of course shows no filling.

The treatment of mucocele of the gall-bladder is by cholecystectomy, which usually presents no great difficulty after the tense organ has been caused to collapse with a trocar and cannula.

In Chronic Cholecystitis.

Chronic cholecystitis goes hand in hand with cholelithiasis, so that the two terms are often used loosely as synonyms. But chronic cholecystitis occurs in the absence of gall-stones. Its surgical pathology is a complex of several diverse entities, the fundamental nature of which is in the main uncertain.

It may occur as a chronic aftermath of frank acute cholecystitis. Here, however, gall-stones are almost always present and playing an essential role in the pathological process. Cholecystectomy is called for.

It may result from a primary low-grade infective process due to the *Streptococcus viridans*, *Streptococcus fæcalis*, various micro-aerophilic streptococci, coliform bacilli, and very occasionally the *Bacillus typhosus*. In these cases the gall-bladder wall is thickened and opaque from subserosal fatty deposition; the cystic gland may be enlarged; in the mucosa flattening of the delicate absorptive villi is present, with œdema and round-cell infiltration. An organ in this diseased state is probably better outside the body than in it; but when the symptoms produced are indefinite and diffuse they are frequently not relieved by cholecystectomy. A cholecystogram showing a non-functioning gall-bladder, therefore, is by no means an indication for operation, for the chronic cholecystitis may be silent and the symptoms complained of be due to pylorospasm, disturbances at the sphincter of Oddi, spastic hyperactivity of the colon, aerophagy and such like states of a functional nature, which are extremely common and very little understood beyond the fact that they are not relieved in the slightest by any type of surgical interference. However, this type of abnormality may well be found at exploratory laparotomy, and under these circumstances few surgeons would hesitate to remove the gall-bladder on the general grounds that it is a diseased organ.

Cholesterosis or "strawberry gall-bladder" may be responsible. This condition was first described by Lord Moynihan early in the century. It is generally regarded as a pathological entity, but its fundamental nature is unknown and its clinical significance most problematical. It would appear to have its foundation in some metabolic disturbance, for the fine villi of the gall-bladder mucosa become laden and fat with deposits of cholesterol and cholesterol esters; but there is no significant invasion by organisms. Boyd (1947) considers that cholesterosis is without doubt a precursor of cholesterol stone formation, but then goes on to say that it is uncertain whether the disease has any clinical importance. These two views would seem to be slightly incompatible.

The strawberry gall-bladder usually gives a more or less normal cholecystogram and is regarded in most quarters as being symptomless.

Various functional disturbances with and without minor degrees of inflammation or degeneration of the cholecyst mucosa are grouped here for convenience, because they are clinically very similar to chronic cholecystitis. European literature, especially the French, contains most of the detailed references to these dystonias of the gall-bladder, and there are numerous descriptions of various methods of investigation, such as radiomanometric studies and duodenal aspiration, which are apparently carried out in France in order to establish a diagnosis of stasis in the

biliary tract (Poilleux and Guy-Albot, 1947; Poilleux and Guillet, 1947). These dystonias are apparently dyskinesias in the region of the cystic duct and the sphincter of Oddi, and French surgeons are prone—probably under the influence of the great Leriche and his school—to endeavour to remedy the disorders by sympathetic ablations, splanchnic and vagal nerve resections and the like. A valvular block at the cystic duct is also described (Caroli, Hepp and Mercardier, 1947), for which the authors recommend cholecystostomy or cholecystoduodenostomy or, apparently in the severe cases, cholecystectomy. I am not in a position to pass an enlightened opinion on these subjects, which seem to be accorded only an uninterested recognition by most Australian surgeons. However, it seems to me, that if the symptoms in any particular case are sufficiently severe to warrant a major surgical assault, then cholecystectomy will bring about a reliable cure when troubles are sited at the cystic duct, and if one is a confident believer in spasms at the ampulla and can diagnose them by manometric tests, then an efficiently performed sphincterotomy is likely to be more certain in its effect than the section of some nerve or other far removed from the seat of the disorder.

This brings us back to the proposition put forward earlier—to wit, that in cases of equivocal biliary disease it is the symptom complex and its severity which are the first and last consideration in a decision to operate. In other words, when special investigations are completed a review of the patient's complaints supplies the deciding evidence.

Migraine.

A word may well be written at this stage on the subject of migraine. Migraine is a fairly common ailment presenting a symptom complex which should be recognizable by all doctors concerned with the treatment of illness, except perhaps dermatologists. It has been my experience that this diagnosis is often missed, and most often by general practitioners who have an enthusiasm for surgery. In some way the abdominal pain and the vomiting of bile seem to suggest some biliary abnormality, so that cholecystectomy is performed, without the slightest benefit to the suffering patient. Headache is not a feature of gall-bladder disease—nor indeed of any other surgical disease that I can think of outside the skull—and it should place one on guard immediately against gross errors of this type. Abdominal pain, when present, is minimal and diffuse, and as often as not probably the result of the strain of vomiting. Abdominal migraine is spoken about occasionally, and I take it to be a sort of cyclic vomiting in adults; in any case it is difficult to see how an attack of this kind could be mistaken for biliary colic by a reasonably astute and careful observer. To those who diagnose migraine and then think wishfully along the lines that the biliary system is in some way causative, I would say that very fair results follow even moderate degrees of therapeutic ingenuity, but surgery is futile.

Acknowledgement.

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THE MORTALITY IN AUSTRALIA FROM MEASLES, SCARLATINA AND DIPHTHERIA.

By H. O. LANCASTER,

School of Public Health and Tropical Medicine, Sydney.

SOME general factors affecting the mortality from the whole group of acute infective diseases, together with pertussis mortality, have already been considered in a previous paper (Lancaster, 1952). The discussion of the mortality from the infective diseases, spread by the nose and throat and affecting predominantly children, is continued here. It was pointed out in the previous paper that it was not possible to give each disease in full detail, but only to discuss some of the points brought out by the official statistics. In studying this group of diseases in Australia, special reference must be made to the detailed account of them from 1788 to 1925 given by Cumpston (1927).

Seasonal Incidence of Infective Diseases.

Most studies on seasonal incidence are from the northern hemisphere. Cumpston (1927) has given seasonal incidence for diphtheria, scarlatina, measles and pertussis by both deaths and notifications, when the latter were available. Here the deaths are used as an index of the incidence, as most of the deaths in this group of diseases occur within a few weeks from the onset. It is convenient to consider the Australian experience for the years 1911 to 1920, summarized by Wickens (1927). Table I has been made from this report on the census. The findings, in brief, are as follows. Pertussis deaths occur throughout the year, but there is an increased number of deaths in the months from September to February, spring and summer. Measles deaths are concentrated slightly earlier than those due to pertussis, and there is a more pronounced seasonal swing, most deaths occurring in the months from July to January. Seasonal variation is less pronounced in the case of diphtheria, the larger numbers of deaths occurring in the months from April to August—that is, in late autumn and winter. Scarlatina deaths occur chiefly in winter and spring, from May to October. The bowel infections—typhoid fever, gastro-enteritis and diarrhœa—have their greatest incidence in the hotter months, from November to April. The difficulties of interpreting such seasonal trends have been discussed by Aycock, Lutman and Foley (1945).

Measles Mortality in Australia.

Cumpston (1927), giving a detailed history of measles in Australia, notes that there is some doubt as to the date of the first introduction of measles into Australia, but that probably the outbreak of 1850 in Victoria was the first. This freedom from infection was due, of course, to the long voyage out from England, during which all susceptibles would have been attacked had cases of measles appeared on board. The long time taken for the disease to spread from Victoria to the other colonies is remarkable, for it reached New South Wales in 1853, Tasmania in 1854 and Queensland in 1857. The first outbreaks occurred in South Australia in 1859 and in Western Australia in 1860.

The annual incidence of measles is rather different in Australia from that in England or in other large populous epidemiological units. In English cities measles appears always to be present, but deaths and notifications are more numerous in alternate years. In some cities this alternation of years of high and low incidences has been a pronounced feature of the statistics for many years, as Stocks (1942) has remarked. In the Australian colonies and States the epidemic waves have been longer, particularly in the small States, Tasmania and Western Australia. This is brought out in Table II, where Cumpston's figures are given for the years 1890 to 1923, and those of *Demography*, the annual bulletin of the Bureau of Census and Statistics, Canberra,

TABLE I.

Deaths of Persons in Each Month Classified According to Causes, Australia, 1911 to 1920.

Cause.	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total Deaths.
Typhoid fever	14·2	14·4	15·1	11·7	9·5	6·3	4·3	3·0	2·8	3·3	5·8	9·8	4,496
Measles	8·2	4·0	2·5	3·7	3·4	4·7	7·5	13·3	14·2	13·9	13·1	11·5	2,403
Scarlatina	7·3	5·6	5·0	5·8	9·8	11·4	9·2	9·0	11·3	10·8	7·7	7·1	675
Whooping cough	10·6	7·3	6·1	5·3	4·7	5·7	6·1	7·2	9·7	12·2	12·1	13·0	3,371
Diphtheria	6·9	5·9	7·6	10·3	12·1	11·0	10·9	8·9	7·4	6·5	6·3	6·0	7,271
Gastro-enteritis	15·3	12·6	11·8	9·9	7·4	4·3	3·2	2·9	2·9	4·8	9·9	14·8	35,521
Meningitis (non-tuberculous)	9·6	7·5	8·1	7·5	7·8	7·5	7·8	9·7	8·7	8·8	8·3	8·6	7,710

are given for the more recent years. This effect would be exaggerated if smaller units than the State were considered.

It is desirable to compare the incidence of measles with the incidence of deaths from congenital malformations and of births of the deaf. In an examination of institutional data in Australia it has been shown that in 1899, 1916, 1924 and 1925, 1940 and 1941 there were excessive numbers of persons born who were later to enter the institutions for the deaf (Lancaster, 1951*a*).

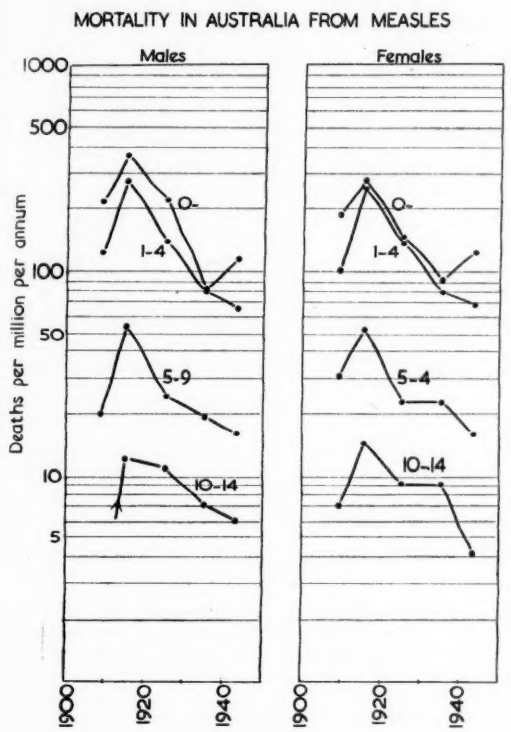

FIGURE I.

The mortality from measles in Australia. Semi-logarithmic grid.[1]

The work of Packer (1950) makes it plausible that measles may act in a manner analogous to rubella in producing congenital defects and deafness. With regard to congenital defects, the official statistics may not be a sufficiently sensitive indicator, but they do not support the thesis that any considerable proportion of the deaths due to congenital defects are due to epidemic causes acting on the mother during pregnancy (Lancaster, 1951*b*).

There is still some room for doubt in the case of the births of the deaf. In New South Wales, 1898, 1915 and 1923 were all years of high measles incidence preceding the high incidence of births of the deaf in the succeeding years. However, there were more measles deaths in 1893 than in 1898, more in 1912 than in 1915, and more in 1920 than in 1923, and yet the year succeeding each of the former of these pairs did not yield any excessive numbers of births of the deaf. Moreover, unpublished work shows that in New Zealand, in 1899, there was an excessive

[1] The three figures of this paper and the figure in Lancaster (1952) have all been reduced to the same scale.

TABLE II.

The Deaths from Measles in Australia by State (Colony) and Year.

Year.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.
1890	7	1	—	1	—	—
1891	10	4	—	—	—	—
1892	2	1	1	—	—	—
1893	730	659	186	261	21	35
1894	143	32	113	28	6	14
1895	2	—	2	2	—	—
1896	4	3	—	—	—	1
1897	3	7	—	—	1	—
1898	510	671	138	54	34	45
1899	209	34	116	27	5	13
1900	10	112	7	2	1	—
1901	37	50	1	5	45	—
1902	107	50	4	235	19	1
1903	16	21	44	5	3	—
1904	21	—	5	—	1	—
1905	29	79	1	—	1	3
1906	17	7	5	1	5	4
1907	90	41	20	5	6	1
1908	32	20	39	14	27	4
1909	11	4	7	1	9	2
1910	99	32	11	—	2	—
1911	44	74	39	16	19	12
1912	371	87	54	15	—	13
1913	51	45	51	20	9	11
1914	19	105	14	11	6	—
1915	324	32	65	37	20	3
1916	73	19	104	20	16	5
1917	30	15	16	11	13	1
1918	76	7	1	1	3	2
1919	8	25	7	9	—	1
1920	189	220	54	15	17	17
1921	39	6	11	15	24	—
1922	16	1	9	4	2	—
1923	138	77	12	1	1	6
1924	36	7	35	—	39	4
1925	30	46	9	45	—	—
1926	90	14	3	—	—	—
1927	20	51	35	7	—	—
1928	162	12	7	2	3	15
1929	66	45	5	8	18	1
1930	100	34	3	7	1	—
1931	29	15	—	1	—	—
1932	14	17	—	1	—	3
1933	45	13	36	11	—	—
1934	34	25	10	8	—	—
1935	83	20	1	—	18	7
1936	22	1	12	—	1	7
1937	8	1	6	—	—	3
1938	—	—	9	—	—	1
1939	100	66	11	11	1	4
1940	22	14	21	30	29	9
1941	8	2	8	1	—	—
1942	130	72	17	8	11	7
1943	13	4	82	3	5	—
1944	4	—	2	4	—	1
1945	5	15	2	1	—	2
1946	37	13	12	15	30	2
1947	7	15	12	2	—	—
1948	49	9	4	8	1	7
1949	23	12	5	5	9	2

TABLE III.

The Mortality from Measles in Australia.

Period.	Sex.	Deaths from Measles per Million per Annum at Ages (Years).								
		0	1	2	3	4	1 to 4	0 to 4	5 to 9	10 to 14
1908 to 1910 ..	M.	222	317	93	55	35	128	148	20	0
1911 to 1920 ..	M.	360	636	290	134	78	289	323	54	12
1921 to 1930 ..	M.	224	313	143	70	35	142	162	24	10
1931 to 1940 ..	M.	84	187	63	45	34	82	84	19	7
1941 to 1945 ..	M.	114	140	41	39	37	66	77	16	6
1908 to 1910 ..	F.	183	246	42	58	43	99	113	31	7
1911 to 1920 ..	F.	277	515	242	165	91	257	272	51	14
1921 to 1930 ..	F.	147	302	107	66	48	132	136	22	9
1931 to 1940 ..	F.	90	173	69	38	32	78	81	22	9
1941 to 1945 ..	F.	120	143	65	24	24	66	78	16	4

number of births of the deaf, but in the preceding year there had been an epidemic of rubella but not of measles.

The epidemic behaviour of measles in Australia is of some interest indirectly, for the epidemic behaviour of measles might be expected to have some resemblance to that of rubella; and there is little epidemiological information about the latter disease in Australia. Both these diseases are virus diseases in which the chronic carrier state is unknown, infection spreading from persons with clinical or subclinical infections, who in a few weeks at most lose their infectivity. Both have a seasonal incidence and confer a relatively lasting immunity. In Australia, as has been seen in the previous paragraph, the measles epidemics are more widely spaced in time than they are in England, and

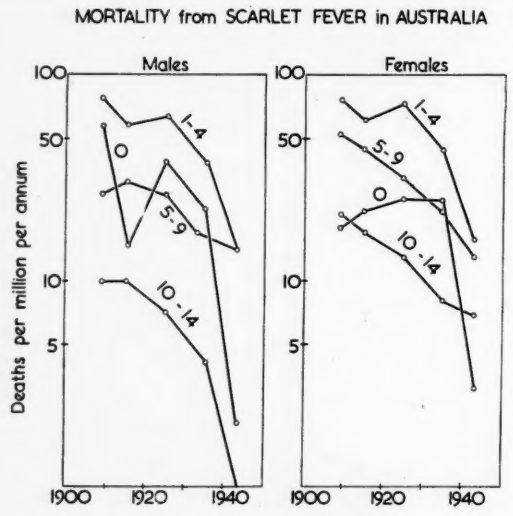

FIGURE II.

The mortality from scarlatina in Australia. Semi-logarithmic grid.

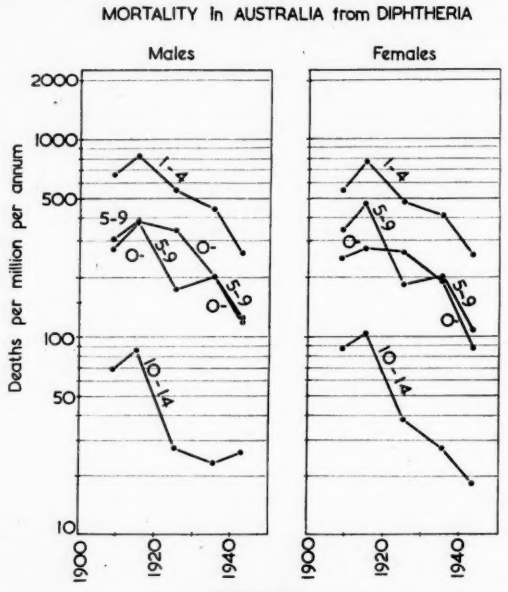

FIGURE III.

The mortality from diphtheria in Australia. Semi-logarithmic grid.

TABLE IV.

The Corrected Notification Rates by Sex and Age (Civilians) in England and Wales (1945) from Certain Infective Diseases.[1]

Ages. (Years.)	Measles.				Diphtheria.				Scarlatina.			
	Notification Rates.[2]		Case Fatality Rates.[3]		Corrected Notification Rates.		Case Fatality Rates.		Corrected Notification Rates.		Case Fatality Rates.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
0–	27·6	28·3	14·4	11·7	30	25	112	72	49	53	17·1	5·6
1–	76·7	75·3	3·0	3·1	91	75	101	108	399	368	3·7	2·5
3–	102·6	103·5	0·6	0·7	188	172	61	59	988	1,007	0·9	0·9
5–	61·7	62·8	0·3	0·4	201	221	43	43	1,055	1,236	0·5	0·8
10–	7·4	8·1	0·7	0·1	113	138	25	18	436	611	0·5	0·7
15 and over	0·4	0·5	1·9	2·3	11	20	30	20	23	33	1·4	3·0
All ages ..	13·6	10·2	1·7	1·6	46	46	45	35	199	182	1·0	1·2

[1] These figures have been taken from "The Registrar General's Statistical Review of England and Wales for the Six Years 1940–1945," Text, Volume I, Medical, Tables XLIII, XLVIII, LXIX.

[2] The notification rates are corrected notifications per 100,000 of persons of the same age and sex.

[3] The case fatality rates are deaths per 1,000 corrected notifications.

one might well expect that the epidemics of rubella would be, too. Under these conditions of widely spaced epidemic waves the disease tends to fall on older persons. This has been the experience with many other diseases; for example, in England smallpox was a disease of early childhood originally. Many thus fail to acquire the infection in childhood, and the females may reach their reproductive period without having become immune. It would seem that this is the factor that makes Australian experience with rubella quite exceptional.

In Table III and Figure I are set out the mortality rates for measles by age and sex for Australia in the years 1908 to 1945. Firstly, the mortality rates may be studied by age. The mortality rates are high in the first year of life, but reach their maximum in the second year and then decline throughout life. After the age of fifteen years but few deaths have been recorded in the period under consideration, and so death rates have been given only for childhood. The increase of the mortality rates from the first to the second year of life is due in part to two factors—firstly, a congenital immunity that tends to be lost towards the end of the first year of life, and secondly the increased opportunities for infection that occur when the child begins to walk. A general discussion of measles immunity may be found in the book by Van Rooyen and Rhodes (1940), from which we may quote the following summary of Halliday's work (1924):

(1) In tenements in congested, often "slum" areas, children are exposed to infection at an earlier age than children in new housing estates.

(2) In the former districts measles is essentially an infant's disease.

(3) In the residential areas schoolchildren are the main victims.

(4) The children of the well-to-do public-school class are affected at a later stage still (14 to 18).

TABLE V.

The Death Rates in England and Wales from Infective Diseases.[1]

Cause of Death.	Deaths per Million per Annum at Ages One to Four Years Last Birthday in the Years			
	1911 to 1920.	1921 to 1930.	1931 to 1939.	1940 to 1945.
Measles	2,409	1,095	749	163
Scarlet fever	289	142	103	17
Pertussis	1,178	857	450	198
Diphtheria	830	531	511	272
Influenza	581	268	128	72
Cerebro-spinal fever	55	41	95	132
Non-meningococcal meningitis	451	187	93	57
Pneumonia *et cetera* and bronchitis	4,250	3,080	1,820	842
Diarrhœa and enteritis	1,182	464	246	124
All tuberculosis	1,522	732	609	464
All causes	15,620	9,396	5,716	3,728

[1] The material of this table has been taken from "The Registrar General's Statistical Review of England and Wales", text volumes for 1932, 1935, 1938 and 1939, and 1940–1945; the figures for non-meningococcal meningitis and for "all causes" come from the volumes for the years 1911 to 1914.

These findings of Halliday come out of an inquiry into mortality in Glasgow. They are of great importance because they show that bad hygienic conditions are associated with infections at an early age. That the delay of infection is of great importance in the control of the mortality can be seen by comparing the case fatality notes at different ages. Such statistics are not thought of sufficient interest to collect in Australia. In Table IV are given the corrected notification rates and the case fatality rates by age and sex in England and Wales for 1945. It is to be noted that the case fatality rates are about four times as high in the first year of life as they are in the second and third years of life. The case fatality rates continue to decline and are quite trivial at ages over five years. The obvious conclusion is that infection must be prevented in the younger children. In England the notification rates attain a maximum in the age group three and four years last birthday according to these figures.

Change in family size has probably had some part in the reduction of mortality from measles in Australia, for as has been pointed out by Halliday (1924) and by Aycock and Eaton (1925), infection of the younger children is very often from their older brothers and sisters. This factor produces a powerful class gradient in the mortality from measles in England and Wales, for in the congested areas there is much mingling of children of all ages. In the residential type of suburb there is less mixing and family size is perhaps smaller, while in the public school classes the older children will probably have their measles while in residence at school and so will not have an opportunity to infect the younger members of their family.

Sex does not play much part in the mortality or case fatality rates. In the English figures the case fatality rates are higher in the first year of life in the male, but in childhood the rates are otherwise practically equal. At ages over ten years, the fatality rates depend on so few cases as to be not very reliable. In the Australian figures of Table III, the mortality rates in the first year of life are higher for the males than for the females, but in the years since 1930 the rates are higher for the females. At ages one to four years, the rates are consistently higher in the males. At ages above five years, the rates are approximately equal.

For any age group over the period of the survey, the mortality is declining. This is shown graphically in Figure I. It is of interest that in the earlier years, 1908 to 1920, the Australian rates were only about one-third of the English rates for children at ages one to four years last birthday (a sufficiently good approximation is to take the mean of the male and female rates in Australia for comparison); but over the war years the English rates were more favourable than the Australian. It was believed in earlier years that there was some under-reporting of the measles deaths in England and Wales. There has probably been little under-reporting in the later years in either country. It is of importance to note that the fall in Australia in the mortality rates from measles occurred in the absence of any improvements in therapy or active measures in prophylaxis.

Comparisons of the Australian figures with the English may be carried out with the aid of Table V.

The Mortality from Scarlatina in Australia.

Cumpston (1927) notes that the first case of scarlatina in Australia was observed in Tasmania in 1833, and that in 1841 cases were observed in Victoria and New South Wales. There is doubt as to when the first cases occurred in other States, but it may be stated that it was many years before definite cases were recorded in them. In the period covered by the present survey, scarlatina had already become a minor cause of mortality even in childhood. The mortality rates of the disease are given by age and sex for the childhood years in Figure II and Table VI. It is seen that it has the maximum mortality in the years one to four. Over the age of fifteen years, few deaths have occurred. The mortality in infancy is less than in the second year of life.

TABLE VI.

The Mortality from Scarlatina in Australia.

Period.	Sex.	The Deaths from Scarlatina per Million per Annum at Ages (Years).								
		0	1	2	3	4	1 to 4	0 to 4	5 to 9	10 to 14
1908 to 1910	M.	57	84	67	96	71	79	74	26	10
1911 to 1920	M.	15	45	68	67	49	57	51	30	10
1921 to 1930	M.	38	53	75	83	39	63	58	26	7
1931 to 1940	M.	22	49	33	38	31	38	35	17	4
1941 to 1945	M.	2	12	19	10	17	14	12	14	1
1908 to 1910	F.	18	61	106	58	80	76	61	53	21
1911 to 1920	F.	22	53	70	60	62	61	55	44	17
1921 to 1930	F.	25	78	69	80	63	73	63	32	13
1931 to 1940	F.	25	64	42	38	32	44	41	22	8
1941 to 1945	F.	3	19	20	14	10	16	13	13	7

TABLE VII.
The Mortality from Diphtheria in Australia.

Period.	Sex.	Deaths from Diphtheria per Million per Annum at Ages (Years).								
		0	1	2	3	4	1 to 4	0 to 4	5 to 9	10 to 14
1908 to 1910 ..	M.	279	730	701	726	567	683	584	318	68
1911 to 1920 ..	M.	381	839	844	904	743	833	776	388	87
1921 to 1930 ..	M.	345	767	577	525	382	564	527	165	27
1931 to 1940 ..	M.	201	452	497	483	336	442	398	201	23
1941 to 1945 ..	M.	120	286	257	261	262	267	233	127	26
1908 to 1910 ..	F.	250	581	664	575	456	570	477	351	88
1911 to 1920 ..	F.	279	758	809	796	804	791	706	470	103
1921 to 1930 ..	F.	263	594	548	442	367	489	445	185	38
1931 to 1940 ..	F.	190	383	457	396	406	411	370	201	27
1941 to 1945 ..	F.	87	307	248	268	203	258	218	109	18

For any age group, the mortality has declined considerably over the period considered. In fact, the rates in the most recent years are only some 20% of the rates holding in 1908 to 1910. This effect cannot be ascribed to improvements in medical science, with the possible exception of the improvement between the period 1931 to 1940 and the latest period 1941 to 1945, possibly partly owing to the sulphonamide drugs and to the antibiotics.

The Mortality in Australia from Diphtheria.

Diphtheria, according to Cumpston (1927), had not appeared in Australia before 1858, although a few sporadic cases of croup had been reported. He has given a history of the disease in Australia up to 1924. In Table VII and Figure III are shown the rates of mortality from diphtheria in Australia. Firstly, the table shows that the disease at any period causes its greatest mortality in the second year of life. The mortality rates decline rather slowly up to the age of five years, but then more rapidly, so that the rates at ages five to nine years are about half those holding at ages one to four years. The disease still causes an appreciable mortality up to the age of fifteen years. In adult life, diphtheria causes few deaths and the rates have not been given here. It appears that the females have a more favourable experience under the age of five years, but that above this age the sexes have approximately equal mortality rates. When the rates for any given age group are compared over the period considered, there has been a fall. This decline has usually been attributed to the immunization campaigns. But the non-specific factors such as improved hygienic conditions, improved nutrition and the coming of the small family group should not be overlooked. For, when the decline in mortality from diphtheria is compared with the decline in mortality rates from other childhood infections, it is seen that its relative decline has been no better than those of measles or pertussis, for which there was no specific treatment or prophylaxis up to the end of the period considered here. The rates of mortality in Australia for children aged one to four years over the years from 1911 to 1945 remained remarkably close to the rates for England and Wales at the same age, as can be seen by comparing the mean of the male and female rates of Table VII with the English rates of Table V. The fall in diphtheria mortality cannot be discussed adequately without an inquiry into the immunization campaigns and an attempt to compute the frequencies of immunized and non-immunized in age-groups, and hence to determine attack rates and case fatality rates by age, sex and immunization experience. Since this point will be investigated shortly in both New South Wales and Victoria there is no need to labour it further here.

Summary.

The mortality in Australia from the infective diseases of childhood has been considered. Some non-specific factors such as the effect of the small family have been mentioned. It is probable that the decline in mortality from pertussis, measles, scarlatina and perhaps even diphtheria has been due to the same set of hygienic factors and not due to specific remedies or active public health measures. Scarlatina has not been a serious public health problem in Australia over the period surveyed here. Measles, pertussis and diphtheria have all been leading causes of death in childhood, and their decline has been an important contribution to the general fall in mortality in childhood that has occurred over the years in Australia. Some comparisons have been made with English statistics, and the lack of continuous and reliable data on notification rates and case fatality rates in Australia has been deplored.

Acknowledgements.

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Reviews.

RADIOLOGY OF THE EAR, NOSE AND THROAT.

"CLINICAL RADIOLOGY OF THE EAR, NOSE AND THROAT", by Eric Samuel, has been received from the publishers, H. K. Lewis and Company, Limited, London.[1] A book of this type has been badly needed, as hitherto most of this class of work has only appeared in special journals. The author has followed an anatomical classification as he considers this allows a readier reference than a purely pathological one. It is laid down that the X-ray examination must be precise and the films of the highest quality, and that reexamination is often necessary owing to individual variations. It is also recommended that examinations should be made as near as possible to operation. Positive findings do not necessarily mean active disease as chronic conditions give similar appearances. Allergic conditions may also cause abnormal dulness, especially in the antra. The author considers that no operation should be undertaken on X-ray evidence alone

[1] "Clinical Radiology of the Ear, Nose and Throat", by Eric Samuel, M.D (London), F.R.C.S. (England), F.F.R. (London), D.M.R.E. (Cambridge); 1952. London: H. K. Lewis and Company, Limited. 10" x 8", pp. 348, with 320 illustrations. Price: 70s.

and that consultation between radiologist and surgeon will give the best results. The presence of pus in the nasal sinuses always indicates the need for radiography. Technique is described and full consideration is given to the developmental anatomy of the paranasal sinuses. Congenital anomalies, foreign bodies and allergic states are described fully and excellently illustrated. The section on frontal mucoceles is very interesting. Cysts and newgrowths receive extensive mention. *Leontiasis ossium*, Paget's disease, dermoids and meningoceles all cause more or less characteristic changes in the X-ray picture, while sarcoma and carcinoma of the antra are quite commonly seen; the latter cause extensive bone destruction. The technique of examination of the mastoid region and temporal bones is presented simply and clearly. The author considers that the pinna should be held forward by adhesive tape during radiography. Here again the developmental anatomy of the region is described in detail. It is surprising how much information can be obtained by careful and meticulous examination. In the consideration of films it is laid down that (*a*) anatomical variations should be noted and whether the cells are of the adult or infantile type; (*b*) if the type is adult, the distribution of cells must be noted and the relationship to the lateral sinus; (*c*) the type of mastoid must be determined—diploetic or sclerotic or mixed. Pathological points are: (*a*) Is the middle ear of normal translucency? (*b*) Are the ossicles visible? (*c*) Is the Eustachian tube visible? (*d*) Are any petrosal cells well defined? (*e*) Are the cells blurred or sclerosed? (*f*) Is any necrosis present? These points should all be covered by the radiologist's report. Pathological conditions are described and illustrated. A useful chapter deals with the post-operative mastoid. The use of X rays in the investigation of diseases of the pharynx and larynx is given much attention. A useful and extensive bibliography is appended to each chapter. This is an excellent work and should prove invaluable to all radiologists.

THE PELVIS IN OBSTETRICS.

THE work of Dr. Howard Moloy and the late Dr. William Caldwell on the morphology of the female pelvis, which began eighteen years ago, is now widely known. The clinical application of their work to the everyday problems of obstetric practice is, however, less generally appreciated. The data accumulated by Caldwell and Moloy led to a synthesis in the form of the recognition of four fundamental types of female pelves, and the acceptance of these four types had the immediate effect on obstetrical thinking of emphasizing developmental, as opposed to pathological, features in the ætiology of variations in pelvic anatomy. The first step towards establishing the clinical value of this work was to calculate the relationships between the determined size and shape of the pelvis (by the use of X-ray pelvimetry) and the actual outcome of labour. Because of the multiplicity of measurable factors this correlation has presented many problems, but has been solved partly by recognizing that certain data are of outstanding importance and that other measurements previously considered important have in fact little clinical significance. Dr. Moloy has successfully correlated the clinical and the morphological in a well-illustrated but inexpensive publication, "Evaluation of the Pelvis in Obstetrics".[1]

The earlier chapters are generously illustrated and describe the classical gynæcoid, android, anthropoid and platypelloid pelvic types. The intermediate or mixed pelvic types are dealt with in less detail and in terms of general principles, the author being obviously determined to prevent the subject from becoming too complex to be clinically helpful. There is an interesting section on the relationship of pelvic type to constitutional habitus.

In view of the extent to which classification of pelvic types by Caldwell and Moloy was based on X-ray pelvimetry, it is pleasing to note the emphasis given to the clinical examination of the true pelvis. The view is convincingly put forward that clinical observations on the character of the subpubic arch, lower part of the sacrum, lateral pelvic walls, ischial spines and sacrospinous ligaments are not only most useful in themselves, but provide, by inference, considerable information on inlet morphology. A useful point is also made in reference to the diagonal conjugate: "The diagnostic significance of the diagonal conjugate diameter", states Moloy, "has been emphasized to such an extent that obvious deviations from the normal in the accessible mid and lower pelvis continue to be overlooked."

Practising obstetricians who are prepared to accept Dr. Moloy's teaching, at least in principle, will find the final sections of the book most interesting and possibly helpful. This section deals with the modifications that may be applied in the use of obstetric forceps, having regard to the pelvic type with which one is dealing and the accepted mechanism of labour for that particular type.

The application of currently available methods of X-ray pelvimetry will not eliminate the need for, or reduce the value of, clinical experience; but a fuller appreciation of pelvic morphology and the use of this knowledge in the correct and judicious use of obstetric forceps will allow the student of today to begin at a point in his knowledge of disproportion, whether at the brim or in the pelvic capacity, which his predecessors reached only after years of painful experience.

[1] "Clinical and Roentgenologic Evaluation of the Pelvis in Obstetrics", by Howard C. Moloy, M.D., M.Sc.; 1951. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 8¼" × 6", pp. 130, with 68 illustrations. Price: £1 3s. 9d.

DISEASES OF THE ENDOCRINE GLANDS.

IN view of recent important advances in therapy resulting from the study of endocrine glands and their secretions, the publication of a comprehensive work entitled "Diseases of the Endocrine Glands", written by Louis J. Soffer with the assistance of co-workers from the Mount Sinai Hospital, has come at an opportune time.[1]

The book is divided into six sections. The first four sections deal with the hypophysis, the adrenals, the gonads and the thyroid, while the fifth and sixth sections are devoted to the thymus and parathyroids, and hypoglycæmia, hyperinsulinism and *diabetes mellitus* respectively. Finally there is an appendix in which laboratory tests of endocrine function are described. In the first five sections the anatomy, physiology and embryology of the various glands are detailed and then the pathology, the clinical features and treatment of diseases affecting these organs are described. In the sixth section, which is written by Henry Dolger, an account of the anatomy and physiology of the pancreas, of the diagnosis and treatment of *diabetes mellitus*, and of spontaneous hypoglycæmia and hyperinsulinism is given. An extensive bibliography is appended to each section.

The views expressed by the author and his co-workers (J. Lester Gabrilove, Henry Dolger and Arthur R. Sohval, who is responsible for the section on the gonads) are sound and based upon extensive experience. Various points are elaborated by reference to detailed case histories. There are 88 illustrations and three coloured plates in the text. Reference is made to the numerous hiatuses in our knowledge of endocrine functions. Controversial questions, such as the existence of a "male climacteric", are discussed in a common-sense and scientific fashion.

In conclusion it may be stated that this informative book will be a useful addition to the library of every practitioner who is interested in the fascinating subject of endocrinology.

[1] "Diseases of the Endocrine Glands", by Louis J. Soffer, M.D., F.A.C.P.; 1951. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 1142, with 88 illustrations and three coloured plates. Price: £8 1s. 3d.

CLINICAL LABORATORY DIAGNOSIS.

IN a span of fourteen years "Clinical Laboratory Diagnosis" by Levinson and McFate has reached its fourth edition as an up-to-date book[2] which is pleasantly set out, giving an outline of physiology, technique, normal and abnormal findings of the various systems in orderly sequence, with excellent cross references where necessary. It is beautifully printed and bound and contains many excellent tables, diagrams and photographs, and thirteen plates, ten of which are coloured.

The book contains much information which is not available in similar books, including chapters on procedure in

[2] "Clinical Laboratory Diagnosis", by Samuel A. Levinson, M.S., M.D., Ph.D., and Robert P. McFate, Ch.E., M.S., Ph.D.; Fourth Edition; 1951. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 1146, with 221 illustrations and 13 plates, 10 in colour. Price: £8 9s.

pædiatrics, tropical diseases, legal medicine and toxicology. Subjects such as blood and plasma bank procedure, sensitivity tests on the latest antibiotics, ACTH tests in Addison's disease, Pilot's method for enumerating eosinophile cells and a brief account of photometric analysis and flame photometry are also included.

Some of the nomenclature such as "methodology", "technologist", "buret" and "metabolor" is strange. Too much space is given to some findings which as yet are only experimental evidence, and too little to some commonly used methods. Also some of the "normals" given are different to local standards and much of the contents is at variance with common local usage. For example, Wright's stain is used, Leishman's is not given; infectious lymphocytosis is described as a part of associated conditions and not as a separate entity; renal glycosuria becomes renal diabetes; a promyelocyte a progranulocyte; a Türk cell a proplasmacyte and a plasma cell a plasmacyte *et cetera*. Some of the plates seem to be poor tinctorial reproductions; others are borrowed from proprietary literature. The type of stain and magnification represented are not stated.

The authors claim that the objective of the book is to present to the student, resident medical officer, practising physician and technician a suitable review of clinical laboratory diagnosis sufficient to meet their general needs. Its unfamiliar phraseology, procedure and normal standards detract considerably from that purpose. However, it is a comprehensive book and should find a place in pathology laboratories as a useful companion to more conservative laboratory manuals.

A TEACHING ATLAS OF GYNÆCOLOGICAL PATHOLOGY.

A MOST valuable teaching atlas of gynæcological pathology has been published by The Williams and Wilkins Company of Baltimore.[1] The authors are Anthony V. Postoloff and David H. Nichols. The atlas consists of one hundred "Kodachrome" slides for projection onto a screen. In a preface the authors state that they have accumulated the illustrations over the years. They include both macroscopic and microscopic reproductions. The text accompanying the slides is divided into six sections, dealing in turn with diseases of the cervix, diseases of the endometrium, diseases of the myometrium, diseases of the Fallopian tubes, benign tumours of the ovary, primary malignant tumours of the ovary and secondary malignant conditions of the ovary. Each section or chapter has a short introductory statement, and then follow short descriptions of the several slides. In some of these short reference is made to clinical histories. The use of slides of this kind is ideal for teaching purposes with either undergraduate or graduate classes. Many teachers prefer to prepare their own slides for demonstration to their students, and this is the ideal method, especially if the students have had an opportunity of seeing and examining the patient before operation and perhaps of being present when the operation was performed. The difficulty in such circumstances is that it takes a long time to build up a complete atlas. In the atlas of Postoloff and Nichols the field of gynæcology is well covered, and this will no doubt appeal to some teachers—surgeons or pathologists.

Notes on Books, Current Journals and New Appliances.

"FAMILY DOCTOR."

THE doctor who is looking for a convincing but readable article for his patients on the "bottle of medicine" fallacy will find it in the August issue of "Family Doctor", the British Medical Association's popular monthly magazine. Attractive as ever, this issue contains much to interest and to instruct (without tears). By a new arrangement the contents are now grouped into four sections. The first section, "Family Reading", has an article on motor-cycle accidents, by the late Sir Hugh Cairns, a delightfully illustrated account of an English country school, a discussion on teaching children to swim, and information on poisonous berries and plants (primarily for the United Kingdom, of course) and on waves (light, sound, X, radio *et cetera*). "Mainly for Mothers" has an article on "Natural Childbirth" and various baby matters. "Medicine and Health" treats sensibly such subjects as blood pressure, sunstroke, travel sickness, care of the feet, the bottle of medicine (already mentioned), dandruff and holiday-time first aid. "Your Home and Family" contains the first of a series of marriage guidance articles, information on bottling and pickling and on hot weather diet, a patient's account of adapting herself to a restricted cardiac income, a "career" article about the non-medical people associated with radiography and radiotherapy, and various regular features. This magazine is well worth boosting and should be in all doctors' waiting rooms. It can be ordered through the Australian agents, Gordon and Gotch (Australia), Limited, for twenty shillings (sterling) *per annum*.

[1] "Atlas of Gynecologic Pathology: Color Film Library and Descriptive Manual", by Anthony V. Postoloff, M.D., and David H. Nichols, M.D.; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9½" × 5", pp. 84. Price: £43 with 100 "Kodachrome" slides.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Cowdry's Problems of Ageing: Biological and Medical Aspects", edited by Albert I. Lansing, Ph.D.; Third Edition; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 1084, with illustrations. Price: £8 1s. 3d.

This edition is intended to be a "progress report" on what has transpired in gerontology since 1942 when the second edition was prepared.

"Foundations of Neuropsychiatry", by Stanley Cobb, A.B., M.D.; Fifth Edition; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6½", pp. 296, with 15 text figures. Price: 32s. 3d.

Intended to give practitioners and students the facts and correlations needed for an understanding of the simpler workings of the central nervous system.

"Principles and Practice of Aviation Medicine", by Harry G. Armstrong, M.D., F.A.C.P.; Third Edition; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6½", pp. 486, with 97 illustrations. Price: 80s. 9d.

In this edition chapters have been revised and four new chapters have been added.

"Patterns of Organization in the Central Nervous System: Proceedings of the Association for Research in Nervous and Mental Diseases, December 15 and 16, 1950, New York", edited by Philip Bard, Ph.D.; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 594, with 268 illustrations. Price: £6 9s.

The volume contains 25 papers chosen from those presented during the sessions. In some instances work hitherto unpublished has been described.

"Biologie, Pathologie und Therapie der Gelenke dargestellt am Kniegelenk", by Arnold Sonnenschein, M.D., D.Ph.; 1952. Basel: Benno Schwabe and Company. 10" × 7", pp. 508, with 219 illustrations. Price: Swiss francs, 54.10.

Deals with the investigation and treatment of lesions of the knee joint.

"Ambulatory Proctology", by Alfred J. Cantor, M.D., with a foreword by Beaumont S. Cornell, M.D.; Second Edition; 1952. New York: Paul B. Hoeber, Incorporated. 9½" × 6½", pp. 580, with 306 illustrations. Price: $10.00.

The first edition was published in 1946; in this edition two new chapters have been added and revisions have been carried out.

"Annual Review of Medicine", edited by Windsor C. Cutting and Henry W. Newman; 1952. Volume III. Stanford, California: Annual Reviews, Incorporated. 9" × 6½", pp. 452. Price: $6.00.

A series of critical reviews covering 17 different subjects.

The Medical Journal of Australia

SATURDAY, AUGUST 23, 1952.

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THE SECRET OF LIVING.

DR. S. W. PENNYCUICK chose Paracelsus as a link between his own subject, chemistry, and that of The Royal Australasian College of Physicians, medicine, when he delivered the Arthur E. Mills Memorial Oration; his choice was a happy one. It has been said of Paracelsus by Victor Robinson in "Pathfinders in Medicine" that it was he who made chemistry the handmaid of medicine. That a chemist should deliver an oration in honour of one who was a leader among practitioners of medicine is therefore fitting. The subject chosen by Dr. Pennycuick, "The Secret of Living", would have appealed to Mills, who showed in his own life that he had discovered the secret, and who set the feet of several generations of students on the path that would lead in turn to their discovery of the same secret if they followed the example set them. Whether Paracelsus discovered the secret of living is a question which would elicit widely differing replies. Robinson writes that there is probably no character in general history, and certainly not one in medical history, about whom such diverse estimates have been expressed as Paracelsus. He names different writers who have described him respectively as an ass, the greatest of Swiss physicians, a loud-mouthed humbug, the best of men, an ignorant vagabond, a sagacious reformer, a worthless charlatan, a high-minded professional man, a parasite hanging on the skirts of science, the torch that lit up the darkness of his age, the chief quack on record and the chief name in medicine. Paracelsus was not modest. He claimed that his views only were correct and his boasting aroused enmity against him. He held that the foundation upon which he wrote was the foundation without which no physician could become established.[1] At the same time, in the presence of the sick, he was (in the words of Robinson) "a changed man; his arrogance and bombast turned to humanity and charity. His heart was naturally noble and at times especially so. He felt himself a father to the patient, which proves him a genuine physician." In trying to form an estimate of Paracelsus we have to remember his early wanderings and his study of alchemy and of the "mysteries" of the occult. At the end of his life he was cut off from the society of other members of his profession. No doubt he had moments of exaltation, if not of ecstasy; according to his philosophy death was the gateway to life, but one cannot imagine that he had a great deal of serenity or happiness, and these will be looked on by most people as essential to the secret of living.

The present purpose is not to determine whether Paracelsus or anyone else learned the secret of living; rather would we try to drive home the lesson set out by Dr. Pennycuick that we ourselves may find out where we stand and also that we may discover whether we can help those around us, particularly the younger group. The reference (admittedly inadequate) made to Paracelsus shows how difficult it may be to determine the position of other people in this matter. Most of those who have discovered the secret of living show it by their general attitude and bearing. These are not the perfect people in whom Dr. Pennycuick says that he is not interested. There is no perfection in the world today. These are the people who display Dr. Pennycuick's four desirable characteristics —they are humble and full of faith, they are tolerant and understanding, they cultivate the precious gift of giving, and they find enjoyment in their daily work. These are the people whom we should wish to imitate. An enigmatical person like Paracelsus may or may not be admirable in more ways than one, but we do not want to imitate him. Dr. Pennycuick describes the secret of living as the secret of getting the most out of life and putting back as much as we get. When in almost the next breath he is prepared to accept the desire of the "modern young person" to get a living and have a good time, this statement must not be separated from its context. The trouble about many young people today—and older ones too—is that they get a living and enjoy themselves and do nothing more. The putting of something back is where many fail.

Every doctor recognizes the fact that the basis of medical practice is the observance of correct medical ethics—the accepted code of medical ethics is the standard by which professional life must be lived. The standard was laid down by Hippocrates and has been accepted ever since by doctors of every age. Every "code" of medical ethics drawn up by bodies such as the British Medical Association is based on the Hippocratic oath. Everyone knows that without a standard of ethics medical practice could not be conducted in a way satisfactory to the public and likely to make medicine more efficient. So is it with life in general. If people desire to have orderly living and wish to promote the welfare of the community they must set up a standard of rules or ideals and seek to put them into effect. And the individual must do the same—he must have a standard to guide him. Whether we call this standard his code of ethics, or his philosophy of life, or his religion does not matter. The standard which will help a man to put back into living something of what he gets out of life is the standard which adopts the "golden rule". It is a standard of service to others and it is not a standard of which one need be

[1] Henry E. Sigerist, "Great Doctors: A Biographical History of Medicine"; 1933. London: George Allen and Unwin, Limited.

ashamed. It does not mean that one who adopts it will be a dour and unhappy person. On the contrary he will be happier than those who know nothing of his standards. He will be what Dr. Pennycuick calls the tolerant and understanding person who cultivates the precious gift of giving. Unfortunately this mental attitude (that is what it is) cannot be handed round like so many savouries at a cocktail party. It should be taught in the early years of life and preferably in the home, by persons who have experienced what they are trying to teach. The subject cannot be further elaborated at the moment. Suffice it to say that those who have learned the secret of living will have no regrets at the end of life. They will be able to say as our tempestuous Paracelsus said:[1] "What is Death? It is that which takes the life away from us. It is the separation of the Immortal from the mortal part. It is also that which awakens us and returns to us that which it has taken from us."

Current Comment.

ROAD TRAFFIC ACCIDENTS IN QUEENSLAND.

THE Government Statistician at Brisbane has issued a bulletin describing the number of road traffic accidents in Queensland for the three months ended March 31, 1952. The bulletin is not concerned with minor accidents reported to the police but not involving death or personal injury, and doing vehicle damage worth less than ten pounds. The number of accidents during the three months' period was 2980, or 108 less than the high total of 3088 for the previous quarter, but 600 more than the number in the first quarter of 1951. The number of deaths, 48, was below the high figures of 74 and 76 recorded in the two middle quarters of 1951. The number of persons injured, 1558, was 316 above the quarterly average for 1950. Twelve motor cycle riders were killed—an increase of nine on the record figure of 21 for the September quarter, 1951, but an increase of two on the figure for the December quarter. Other persons killed in road accidents were six motor vehicle drivers, one pedal cyclist, 17 passengers and 12 pedestrians. Persons suffering from non-fatal injuries included 258 motor vehicle drivers, 332 motor cyclists, 189 pedal cyclists, 536 passengers and 243 pedestrians. The causes of accidents are discussed. Drivers of motor vehicles were held responsible for 50·6% of all accidents. Such accidents, however, include a large proportion which caused damage to property only; in the quarter under review they accounted for only 11 of the 48 deaths and 33% of the non-fatal injuries. The most serious cause of accidents was "excessive speed", which caused four deaths and 80 cases of injury; two deaths and 164 injuries were caused by "inattentive driving". "Dazzled by lights of an approaching vehicle" caused two deaths and 16 injuries, while "not giving right of way at intersection", "intoxication", and "overtaking without sufficient clearance" each caused one death and 47, 29 and 27 injuries respectively. "Not keeping to the left" caused 40 cases of injury. Motor cyclists were held responsible for 8·6 of all the accidents during the quarter; they were responsible for 12 of the 48 deaths and for 15% of the cases of injury. The worst faults of motor cyclists were "excessive speed having regard to the conditions", "inattentive riding", "not giving right of way at intersections", and "intoxication". Pedestrians caused 204 accidents, which resulted in the death of 11 persons and the injuries of 207. Most of the accidents were due to action of adults, but two deaths and 43 cases of injury were caused by children under seven years of age, who were in roadways not under proper supervision of an adult.

Road traffic accidents are not always due to the human element. Defects in motor vehicles caused three deaths and 96 cases of injury; defective motor cycles and defective pedal cycles caused each one death and 16 and 10 cases of injury respectively. Defective steering was the most serious motor vehicle defect. Road conditions were held to be the immediate cause of six deaths and 148 cases of injury. Two persons were killed and 34 injured through horses or cattle straying in the roadway. One death and 30 cases of injury were attributed to weather conditions.

An analysis is made of the time of the day at which accidents occurred. Accidents were at their lowest just before dawn. They occurred most frequently in the middle of the afternoon, reaching the highest level at about 5 o'clock p.m. On the other hand, night time accidents included a much greater proportion of the more serious type of injury than did midday accidents.

A table setting out the number of road traffic accidents in Queensland from 1948 to 1952 shows that the number is constantly increasing. Each of the twelve-month periods named terminates on March 31. The numbers of persons killed for the periods 1948 to 1949 and 1951 to 1952 were 181, 184, 201 and 250. The numbers of persons injured for these periods were 3808, 4612, 5160 and 6392. If these figures are to be compared, they should be considered in relation to the population at risk and the number of motor vehicles on the road.

The Government Statistician is not concerned with the prevention of accidents.

THE AUTOMATIC COUNTING OF MICROSCOPIC PARTICLES.

A DISCUSSION arranged by the National Coal Board[1] in England indicates increasing interest in the problem of counting microscopic particles, which arises in many technological processes such as powder metallurgy and the manufacture of chocolates. To medical workers, the problem of counting red cells and white cells is quite familiar. The development of automatic scanning and counting devices now makes it possible and, indeed, economic to replace the human observer by the machine in some of the more repetitive tasks of hæmatology. The simple enumeration of the total red or white cells appears to be the simplest type of counting problem considered at the discussion; for it appears feasible not only to count automatically, but also to determine the distribution of size of the particles counted. The general method of such automatic counting is to arrange for a scanning beam of light to pass over the field of the hæmocytometer or other counting chamber. Any particles in the illuminated part of the field will cause an electric pulse to be set up. These pulses are recorded and counted electronically, which enables a very high speed of counting to be attained, a matter of thousands per minute. Modifications of the technique allow the particles to be classified by size, so that such machine would be capable of a Price Jones type of count. A number of patents are pending, so that probably suitable models will be available commercially in the near future for use in clinical laboratories.

These procedures will have important effects in hæmatology. Firstly, since it will be practicable for many more cells to be counted than is now customary, much higher accuracy will be attainable. The error of counting is inversely proportional to the number of cells counted. It will be quite practicable to reduce the percentage error from 5% or more for the red cell count to less than 1%. It will be remembered from the general statistical theory, that about 5% of all counts will differ from the true mean by more than twice the percentage error, and not more than 0·3% of counts will differ from the mean by more than three times the percentage error. The increased precision will be of particular value in follow-up studies of the treatment of anæmia or in watching the progress

[1] Quoted by John Hargrave in "The Life and Soul of Paracelsus", 1951. London: Victor Gollancz, Limited.

[1] *Nature*, March 29, 1952, page 518.

of the white cell count under chemotherapy. Moreover, the increased accuracy will lead to a narrowing of the so-called normal range of the various indices, depending on the red cell count, making them more useful. Secondly, the clinical pathologist and his staff, relieved of the tedium of the total counts, will be able to devote more time to morphology and the differential counts. Thirdly, electronic counting will lead perhaps to a more scientific approach to the study of errors in counting. At present there exists in this field of work a rather authoritarian attitude, and arbitrary and even highly improbable criteria are laid down for a good count. The medical student at the outset of his career and the technician, in preference to rejecting practically all of their unsophisticated counts, are forced to reduce the apparent errors either by the selection of squares to be counted or by manipulating the counts obtained. This practice can be shown to fail in its aim of producing more accurate counts and is undesirable from an educational point of view. For a student should record what he sees.

Cost, of course, must be considered. It seems that a counting device can be constructed for much the same price as a good microscope. At an Australian teaching hospital of average size, the economy in wages should pay for the counting device in less than three months.

ULTRASONIC ENERGY IN PHYSICAL MEDICINE.

ALTHOUGH references to ultrasonic energy in medicine have been appearing in European (Continental) journals for a good many years (we mentioned one such article in these columns on August 31, 1940), it has not aroused much interest elsewhere. A good deal of the European work seems to have been uncritical and this may account for the general indifference, though there are features of the subject that are of interest. This will be seen from a broad review of ultrasonics in medical science that was made in a leading article last year[1] and in a more recent reference[2] to the use of an echo-ranging ultrasonic technique in the investigation of the histological structure of organic tissues. Now to help clear the air comes a report[3] on the present status of use of ultrasonic energy in physical medicine. The Council of Physical Medicine and Rehabilitation of the American Medical Association has authorized publication of this report, which is a summary of work performed by a group of investigators of the Mayo Clinic and Mayo Foundation, Rochester, Minnesota. The report describes briefly the general principles of production of ultrasonic vibrations in ultrasonic apparatus and goes on to explain that an ultrasonic wave passing through a liquid alternately compresses and separates its component particles; with high enough intensity the disruptive forces exceed the intermolecular bonds and the medium is actually split apart. This phenomenon, which involves not only the formation of cavities but also their sudden collapse, is called "cavitation". The heating effect of ultrasonic radiation on living tissue brought forward some years ago the therapeutic possibilities of "ultrasonic diathermy", and it is along this line that a good deal of work has been done. European clinicians have also mentioned an "analgesic effect" of ultrasonic radiation. From a survey which they reported in 1950, three of the present group of investigators concluded that, despite enthusiastic claims of some European investigators, enough was not known concerning the physiological effects of ultrasonic energy to warrant its use in treatment of any disease in man; nevertheless, the diagnostic and therapeutic possibilities merited further careful scientific evaluation. Since then the members of the group have sought further evaluation by suitable laboratory studies on living animals. Their method was to ask themselves a series of questions, to carry out experiments in accordance with the questions and to record their conclusions. The report contains details of their experimental work, but we shall quote here only their conclusions. The first of these was that ultrasonic radiation can produce sharply localized heating of living tissues and can cause selective heating of bone cortex and bone marrow as does no other source of energy used thus far for medical diathermy, and further that if ultrasonic diathermy is administered clinically, a wide margin of safety with regard to dosage is necessary because of unexpected variations which frequently arise. The second question and investigation related to the effects of ultrasonic energy on the nervous system. It was found that ultrasonic energy applied in maximal doses over the lower vertebræ of dogs caused paralysis of the hind legs and tail. Furthermore, ultrasonic energy in the amount of 3·0 to 5·8 watts per square centimetre, applied to the mid-thigh, caused complete blocking of, or at least 50% decrease in, the action potential of the intact sciatic nerve in 60% of the dogs studied. Recovery, as indicated by the action potential, was usually complete when the action potential had been only partially decreased by the ultrasonic energy, but recovery was generally incomplete when the amplitude of the action potential had been completely flattened. Direct heating produced a strikingly similar effect on the action potentials. Ultrasonic energy had a much greater destructive effect on the spinal cord than on the sciatic nerve. Further conclusions were that ultrasound of intensities and frequencies employed in physical medicine probably has no effects on conduction in peripheral nerve other than those produced by the rise in temperature, and that it is very destructive to the growing bones of the dog when administered in the manner and doses described in the report. It is recommended that until safe dosages are determined through further investigation, ultrasonic therapy should not be applied to growing bones. Investigation of the effect of ultrasound on tumours revealed no selective action on those treated. There was as much destruction of normal tissues as there was of tumour. It was found possible to cause necrosis at will in the tumours, but never possible to destroy them completely. The final study related to the effect of ultrasound on normal tissues, and it was concluded that very destructive reactions could be produced in tissues, occurring at the point of application of the ultrasonic energy, throughout the pathway of the radiation through the tissues, or at the point of exit. The intensity of the destructive effect varies according to the type and density of the tissues or according to the presence or absence of fascial barriers. The reactions obtained in various animals were similar and varied not with the animal but with the intensity and duration of treatment.

Perhaps the most interesting of the conclusions reached in these studies was a general one of practical importance. It is pointed out that throughout all the studies maximal dosages usually were employed. Yet, except in one study, the dosages did not exceed even half those available on one of the machines which is commonly employed by medical practitioners. Such a machine, employed at maximal dosages, can severely damage many types of tissues; the spinal cord, peripheral nerves, testes, growing bones and hair follicles appear to be particularly susceptible. The present investigators now propose to investigate the effects of smaller dosages and to attempt to find out what the optimal dosages, if any, may be. There may well be a place for ultrasonic energy in physical medicine, but until more exact information is available of the type being sought by the Mayo investigators, its use in clinical practice is far from safe and not easily justified. This view is reinforced by the conclusions reached in another study[1] by H. P. Schwan (a medical practitioner) and E. L. Carstensen (an electrical engineer). The report of this study will be of particular interest to those concerned with the biophysical aspects of the question. It is in fact restricted to the biophysical problem of the production of heat by ultrasound in tissue and the consequences of the manner in which the transformation of sound into heat takes place; the technique is compared with other methods of producing heat in tissue.

[1] THE MEDICAL JOURNAL OF AUSTRALIA, October 13, 1951.

[2] *Ibidem*, June 21, 1952.

[3] *The Journal of the American Medical Association*, February 23, 1952.

[1] *Ibidem*, May 10, 1952.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

A New Technique in the Treatment of Hyphæma.

S. L. JUKOFSKY (*American Journal of Ophthalmology*, December, 1951) gives a preliminary report on the use of streptokinase to lyse blood which has clotted in the anterior chamber of the eye. He reports its use in two cases. In the first case, after injury to an eye, the anterior chamber filled with blood and the tension rose to 48 millimetres of mercury. After removal of a few cubic millimetres of bloody aqueous, 50,000 units of saline solution of streptokinase were injected into the anterior chamber. Within one hour and twelve minutes the blood had practically disappeared from the anterior chamber. Streptokinase was also used injected subconjunctivally to treat a severe subconjunctival hæmorrhage. The blood disappeared in twenty-five minutes.

Hyperpyrexia in the Management of Ocular Inflammatory Disease.

BENNET Y. ALVIS (*American Journal of Ophthalmology*, December, 1951) outlines the use of fever therapy in ocular disease. He states that the physiological effects of fever therapy include dilatation of peripheral arterioles and capillaries, accelerated circulation, increased metabolic rate, increased white cell count, increased phagocytosis, increased pulse and respiration rates, increase in pulse pressure due to lowering of diastolic pressure, increased activity of the thyroid and adrenals, and increased production of ACTH and cortisone. He considers that the ocular diseases most amenable to fever therapy are infectious diseases due to pyogenic organisms, gonorrhœa, syphilis, brucellosis in the acute stage, and non-specific inflammations in the acute or exudative stage, such as keratitis, iritis, uveitis and optic neuritis. Patients with chronic inflammatory processes such as tuberculosis, chronic brucellosis and most virus diseases are not improved by fever treatment. The author concludes that foreign protein therapy and physically induced hyperpyrexia derive their benefit from acceleration of the body's defence reactions. Hyperpyrexia alone, as produced by physical means, will accelerate all the defence reactions more effectively than any pyretogenic injection or inoculation. Hyperpyrexia is the essential factor; it is not a substitute for chemotherapeutic and antibiotic agents, but an adjuvant rendering them more effective. It acts by aiding in eliminating the cause of acute ocular inflammation and by alleviating the harmful inflammatory process itself.

Monocular Diplopia and Concomitant Strabismus.

HERMANN M. BURIAN AND NANCY CAPOBIANCO (*Archives of Ophthalmology*, January, 1952) describe a case of monocular diplopia and discuss its cause. They state that the condition can occur only in patients who have an anomalous retinal correspondence, and this must not be too deeply rooted. Monocular diplopia is due to the fact that in cases of unstable anomalous retinal correspondence the stimulated retinal elements have two spatial values which are simultaneously brought to consciousness. The authors insist that the two images should be designated not as macular and extramacular image, but as normally and abnormally localized image.

Radiation Cataract.

DAVID G. COGAN *et alii* (*Archives of Ophthalmology*, January, 1952) review the clinical findings in 20 early cases of radiation cataract and histological changes in 27 cases of radiation cataract. They state that originally the opacity appeared to consist of a dot usually at the posterior pole, and as this increased in diameter there appeared pepper-like granules and vacuoles scattered about it. The main opacity then developed a relatively clear centre. At the same time granular opacities and vacuoles were found in the anterior subcapsular zone, especially in the pupillary area. These changes appeared to be stationary or slowly progressive. Later, progressive opacification of the cortex occurred, forming eventually a mature and non-specific cataract. Posterior polar cataracts, such as are seen in *retinitis pigmentosa* and the so-called *cataracta complicata*, are most easily confused with radiation cataract. The histological changes consist primarily of failure of the cells at the equator to differentiate into lens fibres and early migration of cells beneath the posterior capsule towards the posterior pole.

Anticoagulant Therapy in Occlusive Vascular Disease of the Retina.

IVAN F. DUFF *et alii* (*Archives of Ophthalmology*, December, 1951) analyse the results of dicoumarol and heparin therapy in the management of occlusive vascular disease of the retina. They review the world literature on anticoagulant therapy in occlusive vascular disease and give their results in 47 patients so treated. Thirty-six patients were admitted to hospital and 11 were treated as out-patients. Four received heparin, 27 heparin and dicoumarol, and the remainder dicoumarol only. From an analysis of the literature and their own cases, the authors are able to conclude that after thrombosis of the central retinal vein, 28% of patients may be expected to regain normal vision; the visual acuity of 19% is unchanged, that of 23% grows worse, and 9% may be expected to have glaucoma. After thrombosis of a tributary vein, 29% may regain normal vision, 20% remain unchanged, 19% grow worse, and 2% have glaucoma. A short term of intensive treatment with heparin appears to produce results as favourable as those obtained with prolonged use of dicoumarol. Treatment must be prompt. The authors state that if they had an occlusion of the central retinal vein, they would prefer to have anticoagulant therapy.

Competitive Action of Miotics on the Iris Sphincter.

KENNETH C. SWAN AND LETA GEHRSITZ (*Archives of Ophthalmology*, November, 1951) conducted experiments which indicated that the combined use of various miotics in maximal doses seemed less effective both in degree of miosis and in hypotensive action than the administration of either eserine or DFP alone. The authors have observed that the intense spastic action of DFP on the iris sphincter and ciliary muscles could be modified and made more tolerable if pilocarpine was administered previously. Using rabbits, the authors were able to show that when 4% pilocarpine solution was instilled into the eyes and then 0·25% eserine solution instilled after the pilocarpine had produced its maximal effect, the miosis so produced was not as great and did not last as long as when eserine was used alone. Similarly it was shown that the action of DFP was reduced by prior administration of pilocarpine. The interference produced by pilocarpine on eserine and DFP was less if the drugs were instilled together. The authors recommend that in cases of acute glaucoma of the narrow angle iris block types, eserine alone will be more effective than eserine and pilocarpine. For chronic types pilocarpine may be used to modify the spastic action of DFP and eserine.

Malignant Melanoma of the Iris.

H. B. STALLARD (*The British Journal of Ophthalmology*, December, 1951) describes his technique for the removal of malignant melanoma of the iris and reports its use in six cases. He describes the clinical types of melanomata of the iris. The first is the nodular type, in which the neoplasm is circular, projects well forward from the anterior surface of the iris, has a fluffy nodular surface and shows thin-walled vascular loops, from which blood may leak into the anterior chamber. Then there is the flat and plaque-like type, in which growth is slow, the neoplasm is avascular, and there is distortion of the iris with early ectropion of the uvea and immobility of the affected part of the iris. Finally he describes a diffuse or "ring" sarcoma, which reduces the depth of the anterior chamber and causes glaucoma; this type, because it infiltrates the ciliary body extensively, is unsuitable for removal by iridectomy. Clinical signs of malignant change occurring in a melanoma which has been previously judged benign are increase in size and vascularity and pigmentation.

Intraocular Acrylic Lenses.

HAROLD RIDLEY (*The British Journal of Ophthalmology*, March, 1952) describes the technique and his results in the use of an artificial lenticulus following cataract extraction. It was decided that the lens must be of material which would not set up tissue reaction in the eye, the size and refractive power of the artificial lens must be determined, and a suitable technique for insertion of the lens must be perfected. It was found that "Perspex" was most satisfactory and least likely to set up irritation in the body. Its refractive index is 1·49 and its specific gravity 1·19. The lens is 8·35 millimetres in diameter and 2·40 millimetres in thickness. The radius of the anterior curve is 17·8 millimetres and that of the posterior curve 10·7 millimetres. A peripheral groove allows ease of handling. Special forceps are used for inserting the lens. The author recommends that extracapsular extraction should be carried out. The pupil should be at least five millimetres wide. Two corneal scleral sutures are used. Very careful and complete removal of

the anterior lens capsule is performed, and after expression of the nucleus the anterior chamber is thoroughly irrigated. If it is decided that the posterior capsule has been completely cleared the lens may be inserted forthwith; if not the eye should be closed and the lens inserted at a second operation. After insertion of the lens a peripheral iridectomy is performed and the sutures are tied. Both eyes are padded for forty-eight hours, and the patient is allowed out of bed on the third or fourth day. The author tabulates his results in 27 cases. He states that with the acrylic lens the refraction is usually within two dioptres of that for the other eye.

The Response of the Retinal Vessels to "Priscoline" in Diabetes Mellitus.

MORTIMER R. CHOLST *et alii* (*American Journal of Ophthalmology*, March, 1952) evaluate the effect of "Priscoline" upon the retinal vessels in cases of diabetes, both with and without diabetic retinal changes, by the method of angioscotometry. Twenty-seven diabetics, whose ages varied from eighteen to sixty-nine years, were studied. The duration of the diabetes varied from six months to thirty years. In 12 cases there were typical diabetic exudates. In the remainder no exudates were seen, although there were other signs of diabetic retinopathy in some. In 12 of the 27 cases, poor widening of the angioscotoma was observed after an injection of "Priscoline". In 11 of these cases exudates were present. In 10 cases good widening of the angioscotoma was observed, and in nine of these no exudates were seen. In the remaining five cases there was a fair widening of the angioscotoma. The authors had previously shown that in patients with pronounced arteriosclerosis there is a poor widening of the angioscotoma following "Priscoline" injection. They believe that the presence of exudates in the retina of diabetics is a manifestation of advanced vascular damage.

OTO-RHINO-LARYNGOLOGY.

Tuberculosis of the Upper Air Passages.

F. C. ORMEROD (*The Journal of Laryngology and Otology*, July, 1951) states that figures have established the fact that there has been during the past twenty or thirty years a steady decrease in the number of tuberculous lesions of the upper respiratory areas until they are becoming relatively uncommon. The mortality from tuberculosis in Great Britain has risen and fallen with deterioration or improvement of the protein food supply. The improved economic state of the population and facilities for earlier diagnosis also have had considerable effect. The improvements in treatment have also played a part in these changes. Immediately after the first World War artificial pneumothorax and phrenic nerve operations had a very beneficial effect on patients with established tuberculosis of the throat. During the same period the incidence of tuberculosis of the larynx at the Brompton Hospital was increasing in spite of these new methods of treatment. Later operations on the thoracic cage, thoracotomy with cutting of adhesions, apicolysis and thoracoplasty, were designed to produce more complete immobility of the lung. From 1930 the technique of these operations improved with a resulting increase in the numbers treated and improvement in the results achieved. This coincides with the great fall in the number of cases of tuberculous disease of the upper respiratory passages and is probably one of the factors causing the decline. Cures were obtained when the lungs were responding to treatment. Complete silence and the application of the galvanocautery were the only local measures that had provided any real curative effects. The advent of streptomycin has provided a means of relieving and frequently curing tuberculosis of the throat far superior to anything that has previously been available. At first the emergence of some streptomycin-resistant strains of bacteria was observed. The addition of sodium para-aminosalicylate has prevented the emergence of these resistant strains. Improvement begins early and is most dramatic in acute cases, especially when there are ulceration and pain on swallowing. The need for analgesics and injection or section of the sensory nerves of the larynx has almost disappeared. The larynx may heal and become normal while the pulmonary condition deteriorates. A series of 54 cases with tuberculous laryngitis is reviewed. In all but seven the patients had bilateral pulmonary disease, and nearly half had cavities. In 26 cases the laryngeal condition was cured after streptomycin therapy. In five others there was apparent cure but some residual swelling. There was great improvement in 15 and slight improvement in three. In only five was there no benefit. The majority of patients attain maximum benefit after having received between 40 and 60 grammes, but others continue to improve up to 90 or 100 grammes. The dose of streptomycin varies between half a gramme every seventy-two hours and one gramme per day. Five grammes of sodium para-aminosalicylate may also be given three times daily for up to six months.

Intracranial Division of the Eighth Nerve for Ménière's Disease.

W. E. GREEN AND C. C. DOUGLASS (*Annals of Otology, Rhinology and Laryngology*, September, 1951) state that a follow-up study was made of 587 patients operated on by W. E. Dandy. Audiograms had been taken before and after operation and at later visits. The operation of partial section of the eighth nerve was adopted to stop vertigo and to preserve the hearing in the affected ear. Of 22 patients with tinnitus and impaired hearing in both ears Dandy selected for operation the ear in which the hearing loss or tinnitus was most pronounced. Only 13 of these bilaterally involved patients were free from vertigo; in 10 the symptoms were unchanged. Of 114 patients who had section of the entire nerve on the side of referred tinnitus, only 42 reported complete relief of their tinnitus. The percentage was a little higher in those patients who had section of the vestibular fibres only. The majority of people, in the case of both complete and partial sections, felt that their tinnitus was unchanged. Hearing was totally lost on the side of a complete section of the nerve. In cases of partial section only the hearing was improved in 9·5%, absent in 13·8%, unchanged in 28·2% and worse in 48·5%. Loss of hearing after operation proceeded in several ways. In 48 cases (7·9%) there was a rather sharp high tone loss following operation, possibly indicating that the nerve fibres concerned lie close to the vestibular portion or that they are more susceptible to trauma. The chief type of hearing loss is one in which there was no change immediately after operation, but a slow decline over a period of years. This was the finding in most of the 46 cases in which worsened hearing was reported after partial nerve section. Facial paralysis due to operative trauma was either transient or permanent. There was a higher incidence of facial palsy in those patients who had complete eighth nerve section. Relief from vertigo has been excellent. It is strongly advised that when surgical therapy is indicated in Ménière's disease, intracranial section of the eighth nerve is the treatment of choice.

Hearing Loss and Nutritional Deficiency.

M. JOSEPH LOBEL (*A.M.A. Archives of Otolaryngology*, May, 1951) reports further studies on the influence of vitamin *A* in certain types of impaired hearing. He states that the incidence of tinnitus and loss of hearing in Bright's disease and in cirrhosis of the liver prompted the making of blood studies on all patients with deafness, tinnitus and vertigo. A wide range of blood chemical essays was carried out. The difficulties encountered in the absorption and utilization of carotene from the digestive tract and the additional work imposed on the liver in the conversion of carotene into two molecules of vitamin *A* were realized, and a preparation for parenteral administration was developed. Results in 300 cases in which this therapy was used indicated an average gain of hearing of 17% to 18%; 51 patients failed to benefit from the treatment; 249 patients showed very definite gains in hearing of conversational tones. Amelioration and cessation of tinnitus occurred in the same proportions. With the use of carotene in the treatment of deafness, 33 of 100 patients treated had a temporary gain of between 20 and 40 decibels. In the remainder the results were equivocal, or there was no appreciable gain at all. No better results were obtained when vitamin *B* complex and carotene were used together. It was concluded that the body could utilize carotene up to a point at which a therapeutic effect was established, but the pharmacological result was lacking. Hepatic disorders could influence the absorption and emulsification of fats and consequently of the provitamin *A*. A significant finding in patients complaining of deafness and tinnitus was the usual lower level of vitamin *A*. Results of trials with a new injectable vitamin *A* preparation ("Anatola", Parke, Davis and Company) hold out promise of its therapeutic value in certain types of deafness. Despite the fact that the hearing loss was not always influenced the accompanying tinnitus was greatly relieved. The injections were given in two millilitre doses intragluteally twice weekly, and *B* complex with a high thiamine content was given intravenously twice a week for two to five weeks.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XXXIV.

INJURIES TO THE EYE IN GENERAL PRACTICE.

INJURIES to the eye met with commonly in general practice may affect: (*a*) the eyelids, (*b*) the globe and associated structures, (*c*) the orbit.

There are four main types of injuries: (i) Mechanical—due to concussion with blunt objects, lacerations, or penetration with large and small missiles. (ii) Chemical—due to acid or alkali. (iii) Due to heat. (iv) Electrical—"flash" keratitis, burns.

Concussion of the eye may follow a punch or a kick. It constitutes one of the hazards of tennis, football, cricket and golf. It is common in vehicular accidents, and may occur more simply by falling against a door knob.

Lacerations are commonly caused by flying fragments of broken glass or metal. The risk of injury by the nail which flies back when mishit by the carpenter places hammering in the list of dangerous occupations. In the playground, the child is exposed to the risk of injury from arrows, shanghais and pointed sticks.

The cornea of the machinist is in constant danger from injury by the stream of small particles from the emery wheel and the lathe. Much more dangerous are the tiny missiles which fly from the head of a hammer when it strikes a hard steel surface. Too often the eyeball is penetrated, a possibility which must never be forgotten whenever injury by a foreign body is suspected. Unfortunately, the discomfort of the original injury may be so trivial as to be completely overlooked, and the first intimation of disaster is pain or loss of vision at a much later date.

Strong acids and alkalis are in use in many different industries, and are likely to cause serious damage to the eyelids and the eye.

Heat burns may be caused by explosions, by direct contact with flame, or by the molten particles thrown off when hot metal inadvertently explodes in the process of casting.

"Flash" keratitis amongst welders is the commonest electrical burn. It may also follow exposure to the flash of a short circuit.

Electric cataract may accompany an electric burn when the point of contact is near the eye.

Examination of the Injured Eye.

Whenever possible the vision of each eye should be recorded before the eye is examined. The patient may be conveniently examined lying on a couch with the illumination supplied by a 150 watt lamp on an adjustable bracket. An examining tray should be kept ready equipped with the following: a 2% fluorescein solution[1] in a dropper bottle (sterile), normal saline (sterile), glass rods, a small bowl of cottonwool swabs, small kidney dish.

If the practitioner wishes to decide whether he should treat an injury to the eye himself or refer it to the ophthalmic surgeon, he must at least be able to examine the cornea and anterior chamber thoroughly. This can be done only by concentrating the light from the lamp onto the cornea by a 13 dioptre condensing lens, and by using a monocular magnifier (loupe) with a magnification of at least eight times. The use of this little instrument is quite easily mastered. It is held between the thumb and forefinger of the left hand whilst at the same time the thumb holds the upper lid up and the middle finger holds the lower lid down. The examiner next brings his eye as close as possible to the loupe and then makes the final adjustment to bring the cornea into focus. With the condensing lens in the right hand at the same time he focuses the light on the cornea.

Unfortunately the difficulties of using the monocular loupe are greatly exaggerated. Once the user realizes that it must be held close to the observer's eye as well as to that of the patient, the main difficulty is overcome. The treatment of injuries to the eyeball must be preceded by an exact diagnosis, and unless the practitioner is prepared to examine the eye in the manner outlined above, he would be well advised to refer to the ophthalmic surgeon all his patients with possible injury to the eye.

The patient with a painful eye approaches the examination with some apprehension, and it is often necessary to reassure him. The supine position is advisable as it avoids all risk of syncope. The examination is conducted most conveniently from the head of the couch.

After a preliminary examination of the outer surfaces of the eyelids, the patient is asked to open both eyes, and the lower eyelid of the injured eye is gently drawn down by the thumb, and the lower conjunctival fornix examined. With the patient looking down the upper eyelid is everted and its inner surface examined. This cannot be done if the patient forcibly closes his eyes or looks up. This manœuvre is performed with little or no discomfort by grasping the eyelashes between the thumb and forefinger of the left hand and then everting the lid over a horizontally placed glass rod which gently pushes the upper edge of the tarsal plate down as the lower edge of the lid is rotated upwards. Any obvious foreign bodies on the cornea or conjunctiva are noticed and a search is made for wounds of either membrane. The shape and size of the pupil are observed and its reaction to light is tested. The anterior chamber, that part of the eye between the iris and cornea, must be carefully examined. Blood (hyphæma) or pus (hypopyon) may be present, the anterior chamber may be collapsed with a black knuckle of iris protruding through a corneal wound, the distortion of the iris forming a pear-shaped pupil, or it may be deepened with the iris flapping like a curtain in the breeze, having lost its support from the dislocated lens. A small black hole in the iris indicates where a foreign body has penetrated, and a search should be made for the wound in the cornea.

Fluorescein is of great value in demonstrating the extent of corneal abrasions, burns and ulcers. By clinging to a small foreign body in the cornea it will often make its discovery more easy. One drop is instilled in the lower fornix, the eye closed for a few seconds, and the excess stain washed away with saline from a dropper. The area of damaged epithelium will stain a vivid green surrounded by the unstained healthy epithelium.

Treatment.

Injuries to the Eyelids and Orbit.

A "black eye" is the commonest sequel to a blow in the eye. Because of the looseness of the subcutaneous tissue, the extravasated blood may spread down to the cheek and across the nose to the other eye. The swelling of the lids may hide more serious injuries. The walls of the orbit may be fractured and air forced from the nasal sinuses into the subcutaneous tissues when the patient blows his nose, leading to emphysema. With a more severe injury the maxilla may be fractured, resulting in depression of the floor of the orbit and displacement of the eye downwards. Even without this occurring the force of the blow may extend to the apex of the orbit and involve the optic canal with subsequent damage to the optic nerve, causing partial or complete optic atrophy and blindness. The swelling of the lids may mask the injuries and it is necessary to palpate the orbital margins carefully, and to open the eyelids gently and make some estimate of the vision.

The black eye and emphysema need no special treatment. If the floor of the orbit is depressed an attempt must be made as soon as possible to elevate the depressed fragment—a task for the plastic surgeon. The damage to the optic nerve is probably caused by hæmorrhage into its sheath and interference is of doubtful value.

Superficial lacerations of the lids need careful suturing only. They may be deep and may extend into the eyeball which must always be examined. The missile causing the damage may come to rest deep in the orbit, in the eyeball or even in the cranial cavity. In horizontal lacerations of the upper lid the levator muscle may be severed from its insertion into the tarsus with a resultant ptosis, the latter being masked by the œdema. Vertical wounds involving the interpalpebral margin need special attention, as do the injuries caused by tearing of the lower lid away from its attachment to the inner canthus. The exact nature of the latter is apt to be overlooked and the torn skin sutured without any awareness of the serious nature of the injury. Its site is between the lower lachrymal punctum and the inner canthus. As a result the lachrymal canaliculus is torn across. The lid must be firmly sutured to the periosteum

[1] Fluorescein drops.

℞
Fluoresceini, grains viii.
Sodii Bicarbonatis, grains xii.
Aquam Destillatum, ad one fluid ounce.

in the region of the inner canthus and some attempt made to reunite the severed canaliculus. The injury is not uncommon and may follow a punch.

The treatment of lacerations of the eyelids is not to be lightly undertaken. Expert treatment promptly applied will usually ensure a good cosmetic and functional result. Haphazard suturing too often results in a disfigurement which is a constant reproach to the surgeon, a danger to the eye, and a source of exasperation to the unfortunate plastic surgeon called upon later on to repair the damage caused by exuberant scar tissue.

The same remarks apply with equal force to burns of any nature involving the eyelids. It cannot be emphasized too strongly that any scarring of the tissues of the eyelids is apt to lead to deformities which are not only unsightly but also a danger to vision. No burn or laceration of the eyelids can be considered trivial.

Injuries to the Conjunctiva.

Lacerations of the conjunctiva usually heal without being sutured. They may mask more serious injuries to the eyeball and the wise practitioner will not accept the responsibility of these cases.

Burns of the conjunctiva of whatever nature may lead to crippling adhesions between the eyeball and the lids, and need the help of the ophthalmic surgeon. Burns with strong caustics because of the absence of pain and of obvious signs of reaction are particularly misleading. After a few days of apparently favourable progress, rapid necrosis and destruction of the cornea occur often with complete loss of vision. Copious irrigation with water or normal saline to dilute the burning agent is carried out as a first-aid measure and the patient referred at once for specialist treatment.

This should be carried out for at least twenty minutes after the instillation of 0·25% "Decicain" drops in aqueous (not oily) solution, to relieve the pain.

Concussion Injuries of the Eyeball.

A concussion injury may follow a blow from any blunt object small enough to penetrate the defence offered by the rim of the orbit. It may follow a punch or a blow with a ball. More simply it may occur by striking the eye on the corner of a box, the corner of the lid of an open desk or the knob of a door. The globe is compressed along its anteroposterior axis, and the force of the blow, transmitted through the cornea and aqueous, squeezes the iris against the lens which is pushed back into the vitreous. The iris may be torn radially with hæmorrhage into the anterior chamber (hyphæma), it may be torn from its attachment to the ciliary body, the ciliary body may be torn away from the pectinate ligament with subluxation or dislocation of the lens, and the retina may become detached with hæmorrhage into the vitreous.

If the blow is sufficiently violent, the sclerotic may give way at its weakest part just behind the corneo-scleral margin and the lens be dislocated outside the eye under the conjunctiva. These are but a few of the injuries which may accompany concussion of the eye. The patient will complain of pain and loss of vision varying from a little blurring to complete blindness of the eye. As the nature of the injury can be determined only by expert examination, these cases should be referred at once to the ophthalmic surgeon. A few of the possible sequelæ are recurrent hæmorrhage into the eye, secondary glaucoma, and after subconjunctival dislocation of the lens, sympathetic ophthalmia.

Foreign Bodies.

The commonest injury to the eye is the impaction of a small foreign body on the cornea, on the conjunctiva or under the upper lid. A piece of dust or ash may blow into the eye. In industry, the injury occurs during grinding, burnishing and turning operations, or whilst hammering. No discomfort may be felt at the time of injury, and the characteristic symptom of "feeling something in the eye", with lachrymation and sensitivity to light, may not arise for some hours. When a patient gives a history of recurrent irritation, with lachrymation, and a little photophobia with free intervals over a period of many days, a corneal foreign body should always be suspected—usually so small as to be found only after a careful search.

A thorough examination is essential. The upper lid must always be everted, and the intruder which may be found nestling in the groove just behind the lid margin, wiped away with a moist pledget of cotton wool. Relief is immediate, the foreign body causing great discomfort as it is dragged backwards and forwards over the sensitive cornea. Sometimes it is necessary to examine the upper conjunctival fornix, a monœuvre which is carried out by pressing the globe back into the orbit with the forefinger through the lower lid, whilst the everted upper lid is pressed back with the thumb of the other hand. This will cause the conjunctiva of the upper fornix to spring into view.

The cornea and conjunctiva are carefully examined for the presence of foreign bodies by the aid of the light and loupe as described above. Unless a loupe is used the observer may mistake pigment spots on the iris for a foreign body on the surface of the cornea. Precipitates on the back of the cornea ("K.P.") may also cause confusion.

If the foreign body is not easily found, the cornea should be stained with fluorescein. The dye will often adhere to the foreign body and render its detection easier. It will adhere to abrasions and ulcers of the cornea and demonstrate their exact extent. Here it is worth while to remember that the symptoms of early conjunctivitis, early iritis, corneal ulceration and the abrasion caused by a foreign body are much the same, and that it is unwise to tell the patient that his trouble is of traumatic origin unless the diagnosis is certain. With an obvious foreign body this is an easy matter. Doubtful cases should be referred for an expert opinion.

The foreign body may have been present for some days, the eye inflamed and a ring of infiltration present in the cornea round the site of the foreign body. Pus (hypopyon) may be present in the anterior chamber. These patients should be referred at once to the consultant.

In every case whilst making the examination the practitioner must have at the back of his mind the possibility that a small fragment of metal has pierced the cornea or sclera and entered the globe. The wound of entry through the cornea may be so small as to be easily overlooked. If through the sclera, it is almost certain to be so. Search of the iris may reveal a small hole which appears black. Observed with the ophthalmoscope it appears red, as does the pupil with the light reflected from the fundus. The history will help and injuries received whilst hammering must always be viewed with suspicion. The foreign body is often sterile and purulent inflammation of the eye is uncommon, although it may occur. The subsequent history is that of destruction of the retina by iron poisoning (siderosis) with blindness of the eye. There is also a risk of sympathetic ophthalmia and bilateral blindness.

It is worth while emphasizing again that the practitioner cannot be too careful in his management of what may appear a trifling injury. The issues are so grave that he must be constantly on his guard when confronted with an apparently trivial injury to the eye, that penetration of the globe by a small foreign body has not occurred.

The Management of Corneal and Conjunctival Injuries.

Unless the practitioner has had special training in the treatment of corneal and conjunctival injuries, he is well advised to refer them to the consultant. This may seem an over-cautious policy, but there are several reasons why it is sound.

1. There is a risk that an intraocular foreign body may be missed.

2. Clumsy attempts at removal of an impacted foreign body increase the size of the abrasion, leading to a much longer convalescence. There is a greater risk of infection, and a greater risk of impairment of vision because of the larger scar.

3. Whilst most of these injuries are of a trivial nature, infections with a virulent organism such as the pneumococcus may lead to the loss of the eye.

Removal of a Corneal Foreign Body.

With the patient lying on the couch a drop of a 5% solution of cocaine is instilled into the lower fornix and the eyes are closed for one minute. The eye should now be sufficiently insensitive to allow the painless removal of a corneal or conjunctival foreign body. Occasionally a second drop may be necessary. A long straight surgical needle may be used to remove the foreign body. It should be sterilized by boiling or by immersion in "Zephiran".

The light is brought as close to the eye as possible and the patient asked to look up at the ceiling. The loupe is held between the thumb and forefinger of the left hand whilst the third finger holds the lower lid downwards, and the thumb the upper lid upwards. The needle is held between the thumb and second and third fingers of the right hand, whilst the fourth finger rests on the patient's forehead to give support to the hand. The support given by the ring finger resting on the forehead steadies the hand and gives it accurate control of the needle. At the same time, the fingers

controlling the lids press gently back on the eyeball to control it. A speculum is quite unnecessary.

The point of the needle is inserted beneath the foreign body which is levered from the cornea. If after removal it does not adhere to the point, the needle is reversed and the foreign body is picked up with the blunt end. Emery tends to fragment, and often has to be removed piecemeal. After removal of the foreign body, the cornea is examined and the site of the abrasion may be found to be surrounded with a brown ring of burnt tissue. If left, it will delay healing. Its removal is not an easy matter, and it is unwise for the practitioner to attempt its removal unless he has had special experience.

The sharp needle is to be preferred to a blunt spud provided it is kept under control as outlined above. With a little practice the fingers and the eye soon become coordinated to master the magnified image of the loupe, unnecessary trauma is avoided and the practitioner can be quite sure that he has removed the whole foreign body and has left a clean abrasion.

If he is not prepared to master this simple technique, he should make no attempt to remove a corneal foreign body himself.

Occasionally a piece of steel will enter the depth of the cornea obliquely without perforating it. If it cannot be removed easily, the patient should be referred to an ophthalmologist.

After-Treatment.—After removal of the foreign body a little 10% sodium sulphacetamide ointment is instilled into the lower fornix. In most instances the patient can return to work at once. He is warned that he may experience some discomfort during the day. This can be relieved by the taking of aspirin.

Not in every case is treatment as simple as this, and the practitioner must decide which patients he can safely retain and which he must refer to an ophthalmologist. The following points will help him to decide.

1. If the injury is recent, the eye quiet, the injury small, and the foreign body completely removed, the patient may resume his usual occupation with instructions to return if the eye does not return to normal within the next two days.

2. Injuries of some days' standing with an inflamed eye should be referred to an ophthalmologist. A ring of infiltration around the abrasion is to be regarded with suspicion. Injuries with coal and wood are particularly dangerous. A line of pus (hypopyon) in the bottom of the anterior chamber, together with an infiltrated ulcer, means that the eye is in grave danger, and the need for expert treatment is urgent.

3. If the patient was hammering at the time of the accident, there is always present the risk of penetration of the eyeball. These patients should be referred for an expert opinion.

Flash Keratitis.

Arc-welders are prone to superficial burns of the corneal epithelium from exposure to the arc. The patient complains of varying degrees of pain with photophobia, lachrymation and œdema of the lids.

On examination, the cornea is seen to have lost its normal lustre, and when stained with fluorescein is covered with a myriad of tiny staining points. Not infrequently, in spite of the patient's confident diagnosis, the real source of the trouble is seen to be a foreign body. The use of 2% "Neo-Synephrine" drops at four-hourly intervals will give relief in the milder cases. Aspirin and cold compresses will be of help in the more severe ones. Cocaine or atropine drops must not be prescribed.

In mild cases the patients can continue at work. In those more severe the patients need protection from the light until the severe pain subsides. Dark glasses are usually sufficient with the patient confined to his home.

The condition causes no permanent damage.

Laceration of the Cornea and Sclera.

Laceration of the cornea and sclera may accompany lacerations of the eyelids and follows the same type of accident. The damage may be confined to the eyeball. Gross injuries are only too obvious with a gaping wound of the corneo-sclera through which the iris and vitreous protrude. With smaller lacerations, the breach to the cornea is followed by a gush of aqueous humour which carries the iris into the wound, distorting the pupil, the prolapsed iris appearing as a small black object in the wound. All cases of this nature should be referred to an ophthalmologist, the eye being covered with a pad and bandage.

The laceration, although penetrating, may not be accompanied by prolapse of iris. Once the outer coat of the eye is penetrated, there is a risk of intraocular infection—acute or chronic—and of sympathetic ophthalmia. The slit lamp must be used in many of these cases to decide whether the wound is a perforating one. There is only one safe rule to follow, and that is to refer every case of linear abrasion of the cornea to the ophthalmic surgeon.

The Therapeutics of Injuries to the Eye.

The treatment of minor injuries to the eye calls for only a few simple preparations. The care of major injuries is the responsibility of the ophthalmic surgeon.

1. *Anæsthesia of the Eye.*—Cocaine is still the best local anæsthetic. The instillation of one drop of 5% cocaine solution usually renders the cornea sufficiently insensitive for the removal of a superficial foreign body. Under no circumstances should cocaine drops be prescribed to relieve pain in an injured eye. If the eye is not covered adequately, the drug rapidly desiccates the corneal epithelium. This desquamates, leaving an extensive area of denuded cornea which is extremely sensitive and painful.

Should the treatment be persisted in, the result may be an extensive ulcer of the cornea which will take some time to heal with the risk of scar formation and diminution of vision.

The discomfort remaining after the removal of a foreign body may be relieved by aspirin. If it does not yield to this simple measure, there may be some complication present necessitating more expert treatment. Occasionally in the presence of a virulent organism the onset of purulent inflammation is dramatic and a hypopyon may appear within twelve hours of the injury.

2. *The Use of Atropine Drops.*—Unless the practitioner has had special ophthalmic experience, he should never prescribe or instil atropine drops. Their use is quite unnecessary after the removal of a corneal foreign body. Even after the instillation of one drop of 1% atropine, the ciliary muscle does not recover its function for some days, causing quite unnecessary inconvenience to the patient. The risk of bringing on an attack of acute glaucoma by the use of atropine in a middle-aged or elderly patient is by no means remote.

3. *Protection of the Eye after Removal of a Corneal Foreign Body.*—In most cases sufficient protection against desiccation of the corneal epithelium after the use of cocaine to remove the foreign body is provided for by the instillation of 10% sodium sulphacetamide ointment.[1] A pad and bandage are rarely necessary, and the patient may resume his normal occupation at once with instructions to return the following day if the eye is still uncomfortable.

These minor injuries to the cornea are the commonest of industrial accidents. Skilfully treated, they should lead to very little loss of time on the part of the worker.

4. *The Use of Penicillin and Other Antibiotics.*—There is no indication for the systemic use of antibiotics in injuries to the eye. Unless used locally, they are valueless in infections of the eyeball, including the cornea. Here they are strictly within the province of the ophthalmic surgeon. The only possible indications for their use are: (i) They can be employed without expense to the patient. (ii) They will not do him any harm. These are both motives of rather doubtful expediency.

If the injuries to the ocular adnexa are such that they demand the use of the antibiotics, they must also demand the advice of the consultant.

KEVIN O'DAY,
Melbourne.

[1] There are several different proprietary preparations of this which are conveniently dispensed in small tubes.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney, Crow's Nest, New South Wales, on April 17, 1952. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital. Parts of this report appeared in the issues of August 2 and August 16, 1952.

Recurrent Attacks of Splenomegaly and Anæmia.

DR. T. F. ROSE showed a boy, aged six years, who had had attacks of severe sharp pain in the left hypochondrium associated with vomiting and pallor at approximately monthly intervals during the twelve months before his first examination at the hospital in February, 1952. The attacks were not associated with jaundice, bleeding or purpuric episodes. The boy had had mild jaundice at birth, but not since. At the age of three years he had been treated at the hospital for diarrhœa. There was no mention in his records of a palpable spleen. He had severe asthmatic attacks due to sensitivity to house dust. A maternal aunt had undergone splenectomy for spherocytosis, but no other family history had been found relating to his splenomegaly and anæmia. When examined in February, 1952, two weeks after an attack of pain, the patient was noted to be pale. The spleen was just palpable, tender and firm in consistency, and moved freely with respiration. The liver was not palpable, nor were there any enlarged lymph glands. He had a hæmoglobin value of 12·8 grammes *per centum*, and a total of 4,500,000 erythrocytes per cubic millimetre, with no spherocytes, reticulocytes or nucleated red cells. The leucocytes numbered 13,500 per cubic millimetre, 49% being neutrophile cells, 13% eosinophile cells, 33% lymphocytes and 5% monocytes. Thus there was moderate neutrophilia and pronounced eosinophilia, the latter being due to his allergic state. A fragility test yielded normal findings. The platelet count and bleeding and coagulation times were normal. The patient was Rh-negative, as also was his mother. As the patient was then well again, it was decided to keep him under observation. On March 20 he had an attack of pain in the left hypochondrium, so severe as to make him cry, followed by vomiting and pronounced pallor. When examined next day he was paler than at his previous visit. The spleen was as before, except that it was much larger. The hæmoglobin value was 11·6 grammes *per centum*, and the erythrocytes numbered 3,800,000 per cubic millimetre. Again no spherocytes were seen. The leucocytosis and relative neutrophilia had decreased, but the proportion of lymphocytes had increased to 54%. A Coombs test yielded negative results. The serum bilirubin content was 0·2 milligramme *per centum*. Ten days after the attack of pain the spleen was barely palpable, the hæmoglobin value and erythrocyte count had risen appreciably, and the leucocyte count had increased slightly, 35% now being neutrophile cells and 41% lymphocytes. It was decided that he had a syndrome characterized by cyclic attacks of a painful sudden enlargement of an already enlarged spleen, mild anæmia and neutropenia. As his attacks of pain were becoming worse, and were due to the sudden distension of the spleen from an unknown cause, it was decided to perform splenectomy. At operation the spleen was found to be enlarged to about twice the normal size. It was very firm and attached to the diaphragm by numerous slender adhesions. No macroscopic infarcts were seen, and no obvious spleniculi were present. The liver was enlarged, but appeared normal. There was no evidence of portal hypertension and no ascites. Dr. Colin Graham reported that microscopic examination of the spleen revealed thickening of the reticulum framework with early fibrosis. The lymphoid follicles were small with early fibrosis, and the walls of many of the smaller arteries were thickened. He commented that the changes looked like the early stages of Banti's disease, but that other conditions such as portal cirrhosis could cause similar histological changes in the spleen secondarily.

Abdominal Surgical Conditions.

Dr. Rose also presented patients who had the following conditions, which will be reported more fully at a later date: strangulated right obturator hernia with unusual pain distribution; appendicitis in a "truly splenic" appendix, causing an abscess in the left upper quadrant of the abdomen; volvulus of the terminal part of the ileum causing gangrene of a loop of bowel, with resection and recovery.

Perthes's Disease of the Right Hip.

In conjunction with DR. L. MACDONALD, Dr. Rose showed a boy, aged thirteen years, who about the middle of 1951 had had an attack of pain in the right groin radiating to the outer aspect of the thigh, with mild pyrexia and limping. At that time he held his right hip in flexion, abduction and external rotation. The right quadriceps muscle was slightly wasted. Active and passive movements of the hip were full and painless. A small infected abrasion was present on the inner aspect of the right knee, and the inguinal lymph glands were slightly enlarged and tender. The findings on X-ray examination of the hips at the time were reported as being normal, but in view of subsequent events a review of the films had shown that even then the epiphysis of the right femoral head was flattened when compared with the left. The texture of the epiphysis was normal. The X-ray appearance was characteristic of very early Perthes's disease. The abrasion was treated with penicillin and bed rest and healed in a week. The pain in the groin and the limp recurred one month later, but the boy did not return to hospital until January, 1952, when the limp was pronounced. He did not look well, but had a normal temperature. Movements of the right hip were limited in all directions, especially internal rotation. The limb was not shortened, but quadriceps wasting was noticeable. The blood sedimentation rate was increased (12 millimetres in one hour). Pronounced Perthes's disease of the right femur was now evident in the X-ray appearances, which showed a flattening and irregular increase in density in the right femoral head, with small areas of decalcification in the proximal part of the femoral neck, the appearances being those of *osteochondritis juvenilis*. The comment was made that actually there was no increase in the density of the bone, the appearances being due to the surrounding decalcification of bone that still had blood supply. The patient was admitted to hospital under the care of Dr. L. Macdonald, who was treating him with traction on a frame, with relief of symptoms. Dr. Rose said that the patient's history showed the rapidity with which Perthes's disease might set in. The X-ray appearances might be normal at first and cause difficulty in diagnosis in the early stages. The raised blood sedimentation rate was interesting. To compare the X-ray films in the early case under consideration with those in a later case in which no treatment had been carried out, he showed the films of a woman, aged forty years, who as a child had never been able to run because of pain in her hips. The condition had never been investigated, but recently she had noticed severe pain in both hips whenever she climbed steps. Examination had disclosed considerable painless limitation of internal and external rotation of both hips. Other movements were normal. Recent X-ray examination had shown typical flattening of both femoral heads, with widening and shortening of the femoral necks and *coxa vara*. The appearances were characteristic of late neglected Perthes's disease, with early osteoarthritis.

Rupture of the Rectum.

DR. KEVIN FAGAN showed a boy, aged seven years, who had been admitted to hospital in March, 1951, with the history that while riding a scooter down a steep incline at high speed he had fallen and been thrown with great force. His buttocks had struck the roadway, and the upturned handle of the scooter had entered his anal canal. It was found that the rectum and anal canal had been torn through the left lateral aspect. The wound extended deeply into the ischio-rectal fossa and buttock under the *gluteus maximus*; the bladder and urethra were intact. Gross fracture of the pelvis had occurred. Both ilia were fractured transversely, and the anterior portions carrying the hip joints were displaced upwards with bilateral sacro-iliac subluxation. At operation the rectum and anal canal were sutured with fine chromicized catgut, and the perineal wound was loosely sutured. At laparotomy the upper part of the rectum and sigmoid were found to be contused, but intact and viable. The bladder was intact. A left inguinal loop colostomy was carried out, it being noted that the sigmoid colon was loaded with fæces distal to the colostomy. Convalescence was stormy and delayed. In April a large abscess surrounding the fractured area with both intrapelvic and extrapelvic components was opened and drained. The child became wasted and hypoproteinæmic, as well as exhausted by pain and gross suppuration. The abscess gradually ceased draining, and no gross osteomyelitis developed. The perineal wound slowly closed, apart from a high ano-rectal fistula, situated posteriorly, the result of the fact that masses of fæces had kept coming down from below the colostomy, forcing themselves through the rectal suture line into the perineal wound. By the end of September, 1951, the patient's general condition had greatly improved, his fistula had closed, and his anal sphincter was demonstrably competent. The colostomy was then satisfactorily closed. Dr. Fagan said that the boy still had a rather ungainly gait, as the result of incomplete recovery from flexor contractures at the knees and hips, and from the abnormal posterior projection of his sacrum; but the condition was slowly improving, and Dr. C. D. Langton, who was looking after the boy from the orthopædic aspect, considered that he would have little if any residual disability.

A second patient shown by Dr. Fagan, a man, aged twenty years, had been injured in a motor-cycle accident in May, 1951. His injuries had included wide separation of the

symphysis pubis and subluxation of the sacro-iliac joint on the right side, with an extensive soft tissue wound which followed the junction of the lower limb and the trunk on the anterior and medial aspects. The anal canal and extraperitoneal part of the rectum were divided in the mid-line, both anteriorly and posteriorly. The bladder, prostate and vesicles were contused, but otherwise intact. There was also a closed fracture of the middle of the shaft of the right femur. With the patient under anæsthesia, a catheter was passed and bright blood and clots flowed from it. Necrotic and heavily contaminated tissue was excised, bleeding was controlled, and the anal canal and rectum were loosely sutured with fine chromicized catgut. The groin wounds were sutured after demonstration of intact femoral vessels, and the perineal wound was left open. After exploration of the peritoneal cavity a left inguinal colostomy was performed, followed by suprapubic cystostomy. Because of the fractured femur, control of the lateral dislocation of the right hindquarter was difficult. The difficulty was overcome by applying Hamilton Russell traction to the lower limbs and lateral compression to the pelvic girdle by suspending the patient by a canvas sling around the pelvis. Paralytic ileus developed, but was controlled with a Miller-Abbott tube and intravenous therapy. The suprapubic cystostomy tube was removed on the tenth day, and the wound healed. A large abscess developed in the upper part of the right thigh and had to be drained. The perineum slowly granulated and healed. It then became apparent that there was little sphincteric mechanism left about the anal canal. In November the scar tissue was excised in the mid-line posteriorly, and the ano-rectal ring was reconstituted with silk sutures. When the wound had healed, sphincteric exercises were carried out by the patient, and in December the colostomy was closed. The patient's bowels acted on the fourth day, and at the time of the meeting he was able to control solid or semi-solid motions, but had partial incontinence of liquid stools. He had negligible disability from his pelvic and femoral injuries.

Carcinoma of the Uterus.

Dr. Fagan's last patient, a married woman, aged sixty-four years, had had a history of bleeding *per vaginam* for eighteen months; in the past six months she had had no normal bowel action, had been passing undigested food *per vaginam* with progressive and intense burning and irritation of the vulva, and had lost 21 pounds in weight. On examination she had been found to be very pale and wasted. The vulva and vagina were extremely excoriated, and small bowel contents were being discharged *per vaginam* in large quantities. Satisfactory pelvic examination was impossible, owing to the tenderness of the vulva and vagina; but a hard mass was palpable *per rectum*, high in the pelvis. A barium meal examination suggested the presence of an ileo-uterine fistula, but the findings were not conclusive. After correction of the anæmia and partial restoration of the body proteins by means of serum infusions, a laparotomy was performed. An advanced carcinoma of the *corpus uteri* was found. This had involved a loop of the lower part of the ileum and obstructed it, and had created an ileo-uterine fistula. The mass involved the mesosigmoid in an easily separated adhesion. It had spread widely in the broad ligaments. Total hysterectomy was carried out, with resection of the terminal part of the ileum and ileo-caecal anastomosis. Separation of the mass from the left ureter was difficult, and it was thought that removal was incomplete at that point. Resection of the involved loop of sigmoid colon was necessary also for complete removal of the growth; but the patient's condition was such that that was not considered practicable. For that reason Dr. Fagan commented that the operation must be considered as palliative in nature. Rapid improvement followed operation. Recovery was delayed by wound suppuration and by phlebothrombosis of the left calf; but the patient was discharged from hospital, well, in February, 1952. Dr. Harold Ham had undertaken to give her deep X-ray therapy.

Retroperitoneal Sarcoma.

DR. F. F. RUNDLE showed a married woman, aged forty-four years, who, six months before admission to hospital, had felt a sudden severe pain in the left upper part of the abdomen. The pain was aching and persisted for about two weeks. Examination at that time revealed a small rounded mass in the upper and left lateral part of the umbilical area. Otherwise no abnormality was found clinically, the fæces contained no occult blood, and radiological examination of the colon and urinary tract showed no lesion. Pain in the region of the lump occurred intermittently during the following months, and she also developed lassitude, a feeling of fullness and wind after meals, and dyspnœa on effort. She lost about half a stone in weight. On her admission to the Wakehurst Wing in December, 1951, it was found that the mass had enlarged considerably and was filling the left hypochondrium and extending also into the epigastrium and left lumbar zone. On pelvic examination a swelling was palpable through the right lateral and posterior fornices. Radiological examination revealed slight bulging of the left hemidiaphragm posteriorly. At laparotomy a large hard retroperitoneal tumour was found in the left upper part of the abdomen; a snippet was taken for microscopic examination. The pelvic mass proved to be the right ovary enlarged to the size of a grapefruit. It was removed. Examination of sections from the ovary and retroperitoneal tumour showed in both the same kind of malignant new-growth, the tissue being composed of small round cells like primitive lymphocytes. The pathologist, Dr. C. Graham, made the following comment: "Taking into consideration the clinical description of the tumours as well as the microscopic appearances, I think the primary tumour is a retroperitoneal sarcoma and the growth in the ovary is a secondary deposit." Dr. Rundle said that subsequent X-ray therapy had been remarkably effective. The abdominal mass had become almost impalpable, and the patient had regained all her lost weight, and felt very well.

Paget's Disease of the Nipple.

Dr. Rundle next showed a married woman, aged sixty-eight years, whose right nipple had been ulcerated on and off for ten years and from time to time had bled slightly. There was no pain or other departure from normal health. Examination of the patient showed superficial ulceration of the right areola, with some destruction of the nipple. In several areas the ulceration appeared to have healed over in part. No lump was palpable in the underlying breast, and no enlarged lymph glands were palpable in the right axilla. In June, 1951, radical amputation of the right breast was performed in the New South Wales Community Hospital. There was some delay in wound healing, but otherwise progress was satisfactory. The specimen and section were presented. Microscopic examination showed a carcinoma growing mainly along the ducts, but in places the cancer cells had escaped outside the ducts. There was one such nodule about one centimetre in diameter at least an inch deep from the surface, where the cancer cells were secreting mucus.

Carcinoma of the Rectum.

Dr. Rundle showed a married woman, aged fifty-five years, who had complained of the passage of blood and mucus in the stools for six weeks. Aching pain in the sacral region had been present on and off for many years. She had not lost weight, and there were no other symptoms. Examination of the patient showed her colour and state of nutrition to be normal. Nothing abnormal was palpable on rectal examination, but sigmoidoscopy showed the edge of a heaped-up bleeding ulcer at a distance of 12 centimetres from the anal verge. At laparotomy, carried out through an oblique incision in the left lower part of the abdomen, a small mass was palpable at the recto-sigmoid junction. No enlarged glands were palpable in the mesocolon. No masses were palpable elsewhere in the colon, and nothing abnormal was felt in the liver. A standard abdomino-perineal excision of the rectum was performed on February 4, 1952. The specimen and sections were shown at the meeting by Dr. Rundle, who said that although the primary lesion was not large, deposits of carcinoma were seen in lymph nodes removed from the level at which the inferior mesenteric artery was ligated. They were further surprised when a large polypus with a very long pedicle prolapsed through the colostomy stoma ten days after the operation. At a subsequent operation the paraaortic lymph nodes were removed *en bloc* up to the superior border of the second lumbar vertebra. The inferior mesenteric artery was doubly tied at its origin, and the inferior mesenteric vein was ligated at the level of the duodeno-jejunal flexure. The whole of the descending colon, the splenic flexure and the left third of the transverse colon were resected, the distal end of the remaining part of the transverse colon being brought out to form a new colostomy to the right of the umbilicus.

Carcinoma of the Male Breast.

Dr. Rundle's next patient, a man, aged fifty-four years, had noticed a lump in the left breast for two months. It was enlarging and the skin over it was itchy, but there was no pain or other symptom. Examination of the patient revealed a hard irregular lump, approximately 2·5 centimetres in diameter, below and lateral to the left nipple. The overlying skin was tethered to the lump, which was also

slightly fixed to the underlying muscle. Many enlarged hard glands were palpable in the left axilla. An intensive course of radiotherapy was given to the left breast and axilla, following which, in December, 1951, radical amputation of the breast was performed in the War Memorial Hospital. Microscopic examination showed a moderately well-differentiated adenocarcinoma of the breast.

Retrosternal Goitre.

A married woman, aged fifty years, shown by Dr. Rundle, had had a goitre for many years, but it had caused her no symptoms until one of the veins on the right side of the neck became very prominent five months before she attended for consultation. There were no other relevant symptoms. No clinical evidence was found of hyperthyroidism or hypothyroidism. Her colour and nutrition were normal. A small, nodular, deeply placed goitre was palpable at the root of the neck, especially on the right side. The right external jugular vein was grossly distended, and the subcutaneous veins were prominent over the *manubrium sterni* and the upper two intercostal spaces on both sides. Radiological examination showed a rounded shadow extending from the neck into the retromanubrial space. In commenting on this patient, Dr. Rundle discussed the pathology of venous obstruction in goitre.

(To be continued.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

VACCINE INSTITUTION.[1]

[*Sydney Morning Herald*, June 18, 1850.]

Report on vaccination for the year 1849, being the third since the establishment of the Institution in Sydney.

Vaccine Institution,
April 19, 1850.

Sir,

It is with much satisfaction that I am in this my third annual report, enabled to announce that without intermission the vaccine virus has been sustained in and transmitted from this Institution to all parts of the colony during the whole period.

2. I beg further leave to state, for the information of His Excellency the Governor, that I have had acknowledgement of the efficacy of the lymph forwarded from hence to Van Diemen's Land, Adelaide, New Zealand and the Sandwich Group.

3. I forward lymph to California, as well as vaccinate those children whose parents will permit me to do so prior to leaving this port.

4. I regret that I am not in a position to state the number vaccinated in the colony, not having returns from the country. I have also to regret the indifference evinced by parents as to their children being vaccinated: in some instances as many as five in one family have been left thus neglected.

5. I have long found it necessary to attend daily at the institution, and am frequently obliged to go to their houses to secure an uninterrupted supply.

6. I am satisfied that the City of Sydney has been more attended to, in respect to vaccination, in the past year than for any other for thirteen prior. At this Institution I vaccinated successfully three hundred and forty-four and distributed four hundred and fourteen charges of lymph: the profession in Sydney, although not favoured by all their returns, have, I am aware, been very zealous in so good a work.

In fine, I may be again permitted to say that the humane intent of the Government in forming this institution has been so far successful.

I have, &c.,
ARTHUR SAVAGE.

The Honorable the Colonial Secretary.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

GENERAL PRACTITIONER GROUPS.

SIR: With reference to "S.P.'s" short note in your journal of the 19th instant, I wonder if you will kindly permit an outsider to make a few remarks without any suspicion of wanting to teach Australians "how to run their country".

Unfurnished residences (offered in advertisements for doctors) hundreds of miles away from any furniture dealers and railway charges being as high as they are, in some instances an easily provoked hospital board, the imposition of a weekly rental of thirty shillings or more for an unfurnished house sometimes with a primitive E.C. system, no travelling expenses when joining in an appointment tantamount maybe to giving one's services free for a fortnight or so and the like can scarcely be considered attractive, and all this has been an eye-opener for one from overseas who has seen much of the world both east and west. Appointments are advertised as "with the right to private practice", which may be utterly misleading, because the nearest chemist is hundreds of miles away like the furniture dealers, and, of course, government medicines in the hospital may not be used for such patients, the alternative being for the doctor to maintain stocks which would scarcely pay for their freight in some cases. In a country of high wages seemingly for all kinds of labour except doctors, it would appear that free hosital treatment for everyone without discrimination or a means test is the wildest extravagance, and any country which is unable to balance its budget but indulges in such extravagance is surely very improvident.

Here, one sees boys of school age earning £4 *per diem* for stacking wool in a shearing shed, cooks in a shearing station paid £40 a week and more, wool pressers "knocking up" £6 to £7 a day, shearers taking £12 or more a day, and so on. The only worker who earns less than the boy who stacks wool is the unfortunate doctor. Education and training are at a pitiable discount.

May I put it this way, sir? If governments levied a charge, say, of five shillings a day on every in-patient and a shilling on every out-patient for each visit, there would be more money in the Treasury to remunerate doctors more reasonably and there would be contentment among a deserving section of the population. These highly paid workers, when asked why they drink or visit the "pub" so often, reply that there is nowhere else to go and nothing else to do. But, of course, the "pub" is on the way to the hospital, and once at the latter place, these men would not resent a small charge. As it is, the "pub" collects the money instead.

It may be profitable for Australia to take a leaf from England's book on this subject before things get out of control.

The tragic thing about free medical treatment is want of its appreciation by those whom it is intended to benefit. I do not think I would be required to elaborate on this point, sir, as it must be the universal experience of doctors.

The strange contradiction or paradox is that dental treatment and spectacles are not free as if both these have no connexion with health and fitness.

Yours, etc.,
B. J. BOUCHÉ, M.R.C.S. (England),
L.R.C.P. (London).

Boulia,
Queensland,
July 27, 1952.

MALARIA IN AUSTRALIA.

SIR: A few cases of malaria are appearing in all States among service personnel who have returned to Australia from the Far East. The strain concerned is exclusively benign tertian, which fortunately is of nuisance and discomfort value only.

Such cases are readily recognized by those who have experienced the clinical manifestations of malaria, but to those unaware or unmindful of this clinical picture, the onset of an acute febrile disturbance, either alone, in association with some mild infection, or in the post-operative course of some satisfactory surgical procedure, may be confusing. One such case was seen recently following a mild knee injury. The diagnosis is rapidly and simply determined by a blood examination.

It is recommended that all medical practitioners refresh themselves with the clinical picture associated with malaria and with the simple laboratory methods of investigation. Attached is a copy of Army Liaison Letter Number 4/1952 from the Medical Directorate, dealing with this important subject, and issued to all serving army medical officers.

Yours, etc.,

F. KINGSLEY NORRIS,
Major-General, Director-General,
Medical Services.

Army Headquarters,
Victoria Barracks,
Melbourne.
July 25, 1952.

MEDICAL LIAISON LETTER NO. 4/1952.

Part II.—Technical: Malaria.

A. Prophylaxis: General.

1. At the conclusion of World War II prevention of malaria was carried out by the following means:

(*a*) Personal anti-malarial measures: Clothing, repellent, use of mosquito nets at night, etc.

(*b*) Drug prophylaxis: Atebrin tabs. (1) (0·1 gm.) daily was the drug in use. Atebrin was more a suppressive drug than a casual prophylactic.

(*c*) Anti-mosquito measures: Mosquito control was carried out by anti-malarial units, e.g., spraying, draining swampy areas, etc. Use of aircraft for the spraying of DDT was an accepted adjunct.

2. There is little doubt that both personal anti-malarial and anti-mosquito measures are still essential and the methods employed have not undergone any great change. The question of the use of atebrin for suppression, however, was the subject of some controversy at the conclusion of the last war; the necessity of a drug for prophylaxis was not in question.

3. Atebrin as a suppressive agent suffered adversely for the following reasons:

(*a*) It produced a yellowish discoloration of the skin and nails.

(*b*) It produced sensitivity in a few individuals—this was mainly in the form of a dermatitis.

(*c*) A break-through of malaria by semi-resistant strains occurred in the Aitape-Wewak area in 1945.

4. Consequent upon this, two other drugs made their appearance with claims outweighing those of atebrin. These were paludrine (proguanil) and chloroquine. Both these drugs are in use today.

(*a*) Paludrine is used by the British Commonwealth Forces in Japan and Korea. One (1) tablet (0·1 gm.) is taken daily during the malarious period. It is a casual prophylactic for MT parasites, but to a lesser extent for BT parasites. A build-up by taking paludrine for one (1) week before entering a malarious area is not essential, but is desirable in the establishment of routine—more important is a carry over period after leaving such an area.

(*b*) Chloroquine is used by the U.S. Armed Forces. Chloroquine, also a suppressive agent rather than a true casual prophylactic, is taken once weekly (1 tab. = 0·3 gm. chloroquine base) during the malarious period. The U.S. Armed Forces in Japan and Korea give this drug to their members each Sunday, during the malarious period, for ease of administration.

5. Subsequent break through or relapse of malaria after cessation of the suppressive drugs is reported to be fairly high with respect to chloroquine. There have been some cases of malaria occurring in troops returning to Australia after cessation of paludrine suppression. The number is not yet determined, but is believed to be very small, and evidence in these cases indicates lax paludrine discipline. Certainly the number of new cases of malaria which have occurred in Australian troops in Japan over the past three years has been very small—only three new cases being reported during that period (until the end of April, 1952). Further investigation is proceeding in an endeavour to produce firm statistics.

6. Prevention of malaria as practised by the A.M.F. is as follows:

(*a*) Personal anti-malarial measures.

(*b*) Paludrine—1 tab. (0·1 gm.) taken orally daily. To be commenced seven days before entering a malarious area and to be continued for 28 days after leaving such area.

(*c*) Anti-mosquito measures both against adult mosquitoes and breeding.

B. Treatment.

7. The best drugs for treatment of the acute febrile attack and prevention of relapse are: (*a*) chloroquine for MT malaria; (*b*) quinine and plasmoquine (pamaquine) for BT malaria.

8. There is still some investigation being carried out overseas to determine a drug that may replace plasmoquine. One of those suggested is primaquine—according to Sir Neil Hamilton Fairley, the results from primaquine are very little better than plasmoquine, and in any case adequate supplies of this drug are not yet available. Plasmoquine is still recommended for use.

9. In the N.G. campaign it was found that (in Sir Neil Hamilton Fairley's experience):

"(*a*) Most of the men who suffered from malaria contracted both BT and MT malaria.

(*b*) Primary fever was associated with MT parasites in the blood and responded well to routine treatment reinforced by intravenous quinine therapy where necessary; and

(*c*) these patients relapsed, after being taken off suppressive drugs, with BT but not with MT malaria, showing that MT malaria had been radically cured."

10. The following comprehensive course of treatment to eradicate both MT and BT infections is recommended by Sir Neil Hamilton Fairley. This is a 15-day course of treatment and will be adopted by the A.M.F. It is as follows:

1st day—4 tablets of chloroquine (each of 0·15 gm. base) initially, followed eight hours later by two further such tablets (total 0·9 gm. base).

2nd day—2 tablets of chloroquine twice daily, morning and evening after food (total of 0·6 gm. base).

3rd day—2 tablets of chloroquine twice daily, morning and evening after food (total of 0·6 gm. base).

NOTE.—In the first three days a total of 2·1 gm. of chloroquinine base is administered, sufficing to produce radical cure of MT malaria.

4th/15th day (inclusive)—10 grains of quinine and 1 tab. (0·01 gm.) of plasmoquine (pamaquine) taken three (3) times daily after food. This is for 12 days.

NOTE.—During this part of the course radical cure of 80-90% of the patients with BT malaria should be effected.

11. Intravenous quinine therapy (10 grains quinine dihydrochloride in 10 c.c. sterile saline solution) should be used whenever necessary, i.e., in the presence of clinical evidence of acute pernicious or cerebral manifestations, or laboratory evidence of hyperinfection by MT parasites.

12. The toxic features which may be caused by plasmoquine (pamaquine) may occur very occasionally. They are:

(*a*) Abdominal discomfort or colicky abdominal pain sometimes associated with nausea and vomiting.

(*b*) Blueness of the lips, lobes of the ears, finger nails, etc., which is transient and due to methæmoglobinæmia.

NOTE.—It is extremely rare for symptoms (*a*) and (*b*) to be severe enough to justify a change of treatment, but if this is considered necessary, plasmoquine therapy should be stopped and a course of chloroquine given for three days as above. Subsequently, one tablet of paludrine (0·1 gm.) should be taken every day for six months.

(*c*) In approximately 1 in every 1000 cases hæmolytic anæmia with hæmoglobinuria has followed the administration of plasmoquine. Clinically the condition is characterized by a syndrome resembling blackwater fever. The onset has been on the 3rd, 4th or 5th day of plasmoquine administration. This syndrome is a definite contra-indication of continuing plasmoquine therapy. Plasmoquine should never again be administered to any individual who has shown idiosyncrasy of this type.

13. It is essential that a patient undergoing this course of treatment should have adequate rest in bed under medical supervision.

14. Finally, it is of the utmost importance to realize that prevention of malaria is a matter of unit discipline, that

carrying out of all anti-malarial measures is essential to success and that the only way to prevent the 10% relapse rate in the treatment of BT malaria is not to contract malaria.

15. While it is the direct responsibility of the Formation Commander to implement all measures designed for the prevention of malaria, it is the direct responsibility of the Medical Service to present as strongly as possible the most efficient advice towards the achievement of this object.

PUBLIC HEALTH ASPECTS OF WATER FLUORIDATION.

SIR: The comments of Dr. H. M. L. Murray on the public health aspects of water fluoridation which appeared in THE MEDICAL JOURNAL OF AUSTRALIA, July 19, 1952, page 105, would give a rather erroneous impression to those unacquainted with the facts of the fluoride-dental-caries relationship.

Dr. Murray suggests that the caries inhibitory properties of fluoride, "a fluorine level of one part per million, occurring naturally in a communal water supply, will reduce the incidence of dental caries in children by 60% to 65%", is without scientific basis. The repetition of this assertion "with complete absence of critical analysis, by one writer after another, in popular and pseudo-scientific journals" does not, necessarily imply that the statement is untrue. The main evidence for this statement is derived from the epidemiological investigations of the United States Public Health Service on carefully selected groups embracing 8576 children by H. Trendly Dean, of whom Weaver says (*Proceedings of the Royal Society of Medicine*, Volume XLI, page 284—the same reference as quoted by Dr. Murray): "In 1938 he [Dean] showed the inverse relationship which exists between endemic dental fluorosis and dental caries prevalence, and during the next few years he, in collaboration with various colleagues, published a long series of papers which left *no room for any reasonable doubt as to the caries inhibitory effect of fluorine.*"

Weaver's investigations in North and South Shields agree with the results of surveys elsewhere, particularly in the United States, but the fact which influenced Weaver to suggest that "the caries inhibitory property of fluorine seems to be of short duration" was that the dental caries experience of the children in the fluoride-free area North Shields was much lower than normally found, for generally the "D.M.F." (decayed, missing and filled) rate is eight to ten for children of that age group using fluoride-free water.

That the caries-inhibitory effects of fluoride extend into adulthood are shown by surveys by McKay (*American Journal of Public Health and The Nation's Health*, June, 1948) and Klein (*Journal of the American Dental Association*, June, 1948), and I have shown elsewhere (*The Australian Journal of Dentistry*, August, 1951, page 251), in a more comprehensive criticism of Weaver's findings, that they are not valid in so far as they support the hypothesis that fluoride exerts a postponing effect on dental caries.

The fact that the use of fluorides in water to partially control tooth decay has been approved and advocated by every group of professional health workers in America, including the American Medical Association and United States Public Health Service, would indicate that there must be convincing evidence to support the inhibitory action of fluoride on dental caries. At the present moment 320 communities in the United States are using artificially fluoridated water, and 290 other communities have approved the installation of the process, facts which show the widespread acceptance of this new means of reducing dental decay.

There is a tremendous amount of evidence available supporting the use of fluoridated water, but the beneficial effects of this measure will be delayed by the presentation of misleading arguments which ignore factual foundation upon which they are based.

Yours, etc.,

NOEL D. MARTIN,
Senior Lecturer in Preventive Dentistry,
University of Sydney.

Dental Hospital,
Chalmers Street,
Sydney.

August 5, 1952.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 26, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	..	11(10)	..	1(1)	..	1	..	13
Diphtheria	4	2	5(4)	1(1)	5(3)	..	..	..	17
Dysentery (Bacillary)	..	2(1)	6(5)	1(1)	2(2)	..	..	..	11
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	6	..	..	13(3)	..	..	..	19
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	1	..	1
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	2(2)	..	..	..	..	..	2
Meningococcal Infection	3(2)	5(3)	1	1	..	1	..	..	11
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	3(1)	4(2)	2	6(4)	..	..	..	..	15
Puerperal Fever	1	..	..	..	..	..	1	..	2
Rubella	..	7(4)	..	..	1(1)	..	..	..	8
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	38(26)	15(7)	17(15)	3(3)	4(3)	7(1)	..	..	84
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	1(1)	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	27(22)	18(5)	21(12)	9(8)	9(6)	3	1	..	88
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Post-Graduate Work.

SEMINARS AT THE ROYAL PRINCE ALFRED HOSPITAL.

THE honorary secretary of the Seminar Group, Royal Prince Alfred Hospital, Sydney, announces that a number of changes have been made in the seminar programme for August and September, 1952, which is now as follows:

August 15: Endocrinology and metabolism section, talk by Professor C. H. Best (Toronto). August 22: Cardiology section, "Heart Disease in Pregnancy". August 29: No seminar. September 5: Endocrinology and metabolism section, talk by Professor E. C. Dodds (London). September 12: Thoracic section, "Antibiotics and Drug Therapy in Pulmonary Disease". September 19: Pædiatrics section, "Acute Poisonings in Childhood". September 26: Hæmatology section, "The Role of the Bone Marrow in Hæmatological Diagnosis".

No alteration has been made in the programme for October, 1952.

These seminars are held in the A2 lecture theatre on Fridays from 1.15 to 2.15 p.m. and are open to all members of the medical profession.

Notice.

SYDNEY INSTITUTE FOR PSYCHO-ANALYSIS.

THE second annual meeting of the Sydney Institute for Psycho-Analysis will take the form of a symposium on the psychotherapy of obsessional neurosis. The discussion will be led by A. T. Edwards, G. A. Lawrance, Andrew Peto, Irene Sebire and Cedric Swanton. The meeting will be held at 8 p.m. on Wednesday, September 10, 1952, in the William H. Crago Council Chamber, British Medical Association House, 135 Macquarie Street, Sydney, and will be open to all medical graduates.

Obituary.

FREDERICK ST. JOHN POOLE.

WE regret to announce the death of Dr. Frederick St. John Poole, which occurred on August 6, 1952, at Adelaide.

Nominations and Elections.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Hartshorne, Brian Herbert, M.B., B.S., 1952 (Univ. Adelaide), 14A Curzon Avenue, Millswood Estate, South Australia.

Sumner, Donald Ernest Wesley, M.B., B.S., 1952 (Univ. Adelaide), 92 Portrush Road, Linden Park, South Australia.

Bade, Ronald Werner, M.B., B.S., 1952 (Univ. Adelaide), Royal Perth Hospital, Perth, Western Australia.

Watson, Patricia Bali, M.B., B.S., 1952 (Univ. Adelaide), 14 Albert Street, Dulwich, South Australia.

Elms, Valerie Lois, M.B., B.S., 1952 (Univ. Adelaide) (qualified 1951), 43 Kingston Avenue, Richmond, South Australia.

Hoile, Douglas Edward, M.B., B.S., 1950 (Univ. Adelaide), 19 Adelphi Terrace, St. Leonards, South Australia.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Walker, Ronald Jeffrey, M.B., B.S., 1952 (Univ. Adelaide) (qualified December, 1951); O'Connor, John Desmond, M.B., B.S., 1952 (Univ. Adelaide) (qualified December, 1951); Hughes, John Burnell, M.B., B.S., 1952 (Univ. Adelaide) (qualified December, 1951); Tarlton-Rayment, James, M.B., B.S., 1952 (Univ. Adelaide) (qualified December, 1951); Barnes, Robert, M.B., B.S., 1948 (Univ. Adelaide).

Diary for the Month.

AUGUST 26.—New South Wales Branch, B.M.A.: Ethics Committee.
AUGUST 27.—Victorian Branch, B.M.A.: Council Meeting.
SEPT. 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 3.—Western Australian Branch, B.M.A.: Council Meeting.
SEPT. 5.—Queensland Branch, B.M.A.: Jackson Lecture.
SEPT. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 12.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 15.—Victorian Branch, B.M.A.: Finance Subcommittee.
SEPT. 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital: all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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